THE SENSIBILITY OF THE ALIMENTARY CANAL

ARTHUR F. HERTZ

OXFORD MEDICAL PUBLICATIONS

THE SENSIBILITY OF THE ALIMENTARY CANAL

THE GOULSTONIAN LECTURES ON

THE SENSIBILITY OF THE ALIMENTARY CANAL

DELIVERED AT
THE ROYAL COLLEGE OF PHYSICIANS
ON MARCH 14, 16 & 21, 1911.

BY

ARTHUR F. HERTZ, M.A.,M.D. (Oxon.), F.R.C.P.

ASSISTANT PHYSICIAN AND PHYSICIAN IN CHARGE
OF THE DEPARTMENT FOR NERVOUS DISEASES, GUY'S HOSPITAL

LONDON

HENRY FROWDE
OXFORD UNIVERSITY PRESS

HODDER & STOUGHTON
WARWICK SQUARE, E.C.

1911

PREFACE

With the exception of a short addition to the chapter on hunger, made in accordance with a suggestion of Sir William Allchin, and the references to the work of Weber, Steinhäuser, Glücksmann and Kast and Meltzer, this book is a reprint of the Goulstonian Lectures, as they were delivered in March 1911 before the Royal College of Physicians and published in *The Lancet* in April and May 1911. A division into chapters has been substituted for the original division into lectures.

A preliminary account of some of the observations recorded in these lectures was published in the *Journal of Physiology*, xxxvii, 481, 1908, and in a somewhat fuller form in the *Guy's Hospital Reports*, lxiv, 393, 1910.

I desire to offer my sincere thanks to the authorities of the Royal College of Physicians for the honour they conferred upon me by their invitation to deliver these lectures. I also desire to express my gratitude to my co-workers, whose names appear in the text, as without their help the investigations, upon which these lectures are based, could never have been carried out, and to Dr. C. H. Rippmann for reading the manuscript and for many helpful criticisms.

ARTHUR F. HERTZ.

1 Weymouth Street, W.

May 1911.

CONTENTS

INTRODUCTION

No branch of medicine has made more rapid strides in the last fifty years than that which deals with diseases of the alimentary canal. 'Exact physical information,' wrote Brinton in 1858 concerning pathological processes in the stomach, 'is almost denied us. The aids to diagnosis afforded by auscultation in the diseases of the thoracic viscera, and by chemistry in those of the urinary apparatus, scarcely find any parallel in the maladies of an organ which executes its work without perceptible sound or movement, and only dismisses its products from the body after a complex series of changes and admixtures.' But Brinton's admirable pathological and clinical investigations resulted in the production of a series of 'Lectures on the Diseases of the Stomach', which can be read even to-day with interest and profit.

No further advance was made until fifteen years later, when, after Kussmaul had introduced lavage into gastric therapeutics, von Leube extended the use of the stomach-tube to diagnosis as well as treatment. A method was now available for studying diseases of the stomach, the importance of which was comparable to that of examining the urine in diseases of the kidneys. In the last few years Adolf Schmidt has shown that the examination of the fæces can yield equally valuable information with regard to disturbances in intestinal digestion.

Just as our knowledge of the chemical functions of the alimentary canal was revolutionized in the latter part of the nineteenth century by the introduction of test-meals, so in the present century has our knowledge of its motor functions in health and disease undergone a complete revision as a result of investigations with the X-rays.

Greatly as we are indebted to these new methods of examining patients suffering from diseases of the alimentary canal, medicine owes perhaps even more to the surgeons,

who have founded a true pathology of the living and have enabled us to recognize that there is a definite anatomical cause for many well-recognized groups of symptoms, which had hitherto been regarded as functional in origin.

The use of the stomach-tube and the X-rays and the frequency of abdominal operations for diseases, which were formerly regarded as quite outside the province of the surgeon, have taught us the true significance of many of the symptoms, which were universally misinterpreted until quite recent times. But although the significance of certain symptoms is now clear, their actual cause is in many instances as obscure as ever. With the help of our modern methods of research, it should be possible to throw light upon some of these obscurities. Pain is probably the most common as well as the most trying of all symptoms, and it is to an attempt to elucidate the causes of pain and other abnormal sensations in diseases of the alimentary canal that these lectures are devoted.

It is clearly impossible to understand the origin of pathological sensations so long as the degree of sensibility of healthy organs to various stimuli remains unknown. Comparatively little experimental work has been done on this subject, most of what has been written on visceral sensibility having been founded on theoretical considerations. I have for some time been experimentally investigating the sensibility of the alimentary canal, and I shall first consider what conclusions can be drawn from our observations on the sensibility of the œsophagus, stomach and intestines to tactile, thermal and chemical stimulation. I shall then proceed to the discussion of the sensations of fulness, emptiness and pain. Most of my work on the sensibility of the alimentary canal under physiological conditions has been done in co-operation with Messrs. F. Cook, E. G. Schlesinger and G. Marshall, and when I refer to ' our ' observations, I mean the observations made with these gentlemen, unless the contrary is specially indicated. For help in certain investigations, I am greatly indebted to Mr. H. W. Barber, to Mr. K. H. Digby, to Dr. William Hill and Mr. J. W. Harrison, to Dr. W. Johnson and to Dr. M. S. Pembrey.

CHAPTER I

TACTILE SENSIBILITY

When chemically inert substances at the body temperature are swallowed, no sensation is produced after they leave the pharynx, and no indication of their passage through the stomach and intestines is felt until fæces enter the rectum immediately before defæcation.

The sensibility of the œsophagus to tactile stimuli was tested by pouring thick fluids at the body temperature down an india-rubber tube, the lower end of which was situated at various points in the œsophagus ; this was only done after the tube had been left in position for a sufficient period for the individual to become accustomed to its presence. He never experienced any sensation. In conjunction with Dr. Hill and Mr. Harrison experiments were made with an œsophagoscope, which had a slit down one side of it. A metal bulb in a long holder was warmed to the body temperature and then moved along the mucous membrane of various parts of the œsophagus through the slit in the tube : nothing was felt. It was found that the whole pharynx was sensitive, the disappearance of tactile sensibility occurring abruptly at the upper border of the cricoid cartilage, which is generally regarded as the anatomical separation between the pharynx and the œsophagus, although there is no sharp dividing line at this point in the structure or nerve-supply of the mucous membrane.

The insensibility of the gastric and intestinal mucous membrane to tactile stimuli was proved by the absence of any sensation, when fluids at the body temperature were introduced through a tube by the natural passages in normal individuals and by the artificial openings in patients on whom a gastrostomy or colostomy had been performed.

The sensibility of the rectal mucous membrane was

investigated with Dr. Johnson by stroking the part seen through a rectal speculum with a flat metal instrument, which had been warmed to the body temperature. No sensation was experienced until the anal canal was reached, the mucous membrane of this proving to be very sensitive.

We may thus conclude that *the alimentary canal from the commencement of the œsophagus to the junction of the rectum with the anal canal is completely insensitive to tactile stimulation.*

CHAPTER II

THERMAL SENSIBILITY

Quincke,[1] Neumann[2] and Roux[3] have published observations, which appear to indicate that the stomach is sensitive to thermal stimulation, a conclusion which L. R. Müller[4] and Becher[5] were unable to confirm. In 1846 Weber[6] observed that a cold enema produced no sensation after it had passed the anus; the latter, however, was sensitive to cold. On the other hand Head, Rivers and Sherren[7] in 1905 found that ice-water produced a sensation of cold and water at 50° C. an uncomfortable or hot sensation, when introduced into the intestine of patients on whom a colostomy had been performed.

Our results in the case of the stomach are in opposition to those of Quincke, Neumann and Roux, and agree with those of Becher and Müller, so far as the latter go. We found that the introduction of ice-cold water and of hot water at any temperature between 40° and 50° C. into a normal stomach produced no sensation of any sort. We suggest that Roux's results were due to his having employed an ordinary stomach-tube. When hot or cold water is passed through such a tube, its outer surface rapidly becomes hot or cold, so that the thermal stimulus can act upon the œsophagus. We have used a double tube, an india-rubber tube 5 mm. in diameter being introduced into an ordinary stomach-tube. With this arrangement three or four ounces of water can be passed down the inner tube before the outer surface of the stomach-tube becomes definitely hot or cold. Accordingly we have found, after passing the double tube into the stomach, that no sensation of heat is produced until more than three or four ounces of hot water are poured down, an ill-defined sensation of heat being then experienced; this is due to conduction of the heat to the œsophageal wall,

as the observations on gastrostomised patients to be presently described show that much larger quantities of hot water can be introduced without giving rise to any sensation. As much as half a pint of ice-cold water can be poured through a double tube into the stomach without a sensation of cold being felt.

These observations made it probable that the sensation felt when hot and cold fluids are swallowed is produced in the œsophagus. This was proved to be the case by introducing the tube a distance less than 40 cm. from the teeth, so that its end was in the œsophagus a short distance above the cardiac orifice of the stomach. On now pouring down water at 0° and at 40° or 50° C., a sensation respectively of cold and heat was produced after a latent period of between a half and one second, its character and indefinite localization deep in the epigastrium being exactly the same as when the fluid is swallowed in the ordinary way. On now pushing the tube into the stomach and pouring hot and cold fluid down, no sensation was produced. On four occasions hot and cold water having been poured into the stomach without producing any sensation, the tube was pulled up so that its lower end did not quite reach the cardia ; the temperature sensation was at once felt. X-ray observations showed that the point in the epigastrium where the temperature sensation was felt was generally within an inch of the position of the lower end of the œsophagus, as projected on the anterior surface of the body.

In conjunction with Dr. Hill and Mr. Harrison, I found by means of cold and hot metal bulbs, passed through an œsophagoscope so as to touch the mucous membrane of a normal man in various situations, that the whole œsophagus is sensitive to thermal stimuli, and that the power of localization is extremely accurate, particularly in the case of heat.

Some observations made on a young woman, who had had a gastrostomy performed for a fibrous stricture of the œsophagus, which followed the swallowing of strong hydrochloric acid, and on three men, who had undergone the same operation for epithelioma of the œsophagus, confirmed our belief that the gastric mucous membrane is completely

insensitive to thermal stimuli. Hot and ice-cold fluids were poured through a catheter, which was passed through the gastrostomy tube, this double-walled channel being used so as to prevent the skin surrounding the opening from being warmed or cooled. It was found that no thermal or other sensation was experienced, however large a quantity of the fluid was introduced.

On paying accurate attention to the sensation produced after drinking a mouthful of very hot or cold fluid, it was now recognized that the sensation in the epigastrium began about three seconds after the swallowing act and lasted for about three seconds. On simultaneously listening to the epigastrium it was found that the second deglutition sound, which we had previously shown [8] begins immediately after the last trace of food has entered the stomach, corresponded with the moment when the sensation was disappearing. Hence the period during which the sensation is felt must be that in which the fluid is collected in the lower end of the œsophagus and is slowly passing into the stomach. We may therefore conclude that the feeling of heat and cold experienced when hot and cold fluids are drunk, and until now almost universally ascribed to the stomach, really originates in the lower end of the œsophagus; it lasts for a period which coincides with that during which the fluid is passing through the cardiac orifice into the stomach.

James Mackenzie [9] ascribed the sensation of heat and cold in the epigastrium after drinking hot and cold fluids to reflex vascular changes in the skin of that area, and he believed that the skin is actually the seat of the sensation. We have disproved this theory by placing ice over the skin of the epigastrium so that a maximal sensation of cold was produced; ice-cold water was then poured into the œsophagus through a tube, and immediately a sensation of cold was felt in a situation deeper than that simultaneously felt as a result of the contact of the ice with the skin. On another occasion the skin of the epigastrium was warmed by a flask containing water at 48° C., and water at the same temperature was poured through a tube into the lower end of the œsophagus. A sensation of heat deeper than that felt in

the skin was produced. Suggestion was excluded by blindfolding the individual and pouring hot and then cold water down without saying which was to be done first; the temperature was recognized immediately in both cases.

In a woman with severe bulbar paralysis, in whom no sensory or motor disturbances were present in the limbs or trunk, Dr. Johnson and I found that hot and cold fluids poured into the œsophagus produced no sensation. It is therefore probable that thermal sensations in the œsophagus are conveyed to the central nervous system by the vagi and not by sympathetic fibres.

Since the preliminary publication of our investigations on the sensibility of the colon to temperature[10] they have been confirmed by Zimmermann;[11] they agree also with the old observations of Weber, but not completely with the recent ones of Head, Rivers and Sherren. By introducing hot or cold water through a double tube into the colon of four healthy individuals, we found that the mucous membrane of the large intestine is generally as insensitive to temperature as is that of the stomach. A double tube is required, as otherwise heat or cold is felt in the sensitive anal canal, and it is then difficult to be sure whether the sensation is present elsewhere as well. We have used for the purpose an ordinary rectal tube, through which had been passed a small india-rubber tube just large enough to fit tightly in the round opening at its end. We obtained the same result in six patients, on whom colostomy had been performed for stricture of the rectum, when hot and cold fluids were introduced through a tube into both the proximal and distal parts of the colon. In one normal individual and in two patients a very slight and ill-defined thermal sensation was produced on a single occasion with hot water and in one patient with cold water, although in a number of additional observations on the same individuals no sensation was experienced. The exposed mucous membrane in colostomy cases is invariably quite insensitive to heat and cold, as Steinhäuser[12] was the first to show eighty years ago.

With the aid of a rectal speculum Dr. Johnson, Mr. Barber and I found that a cold or hot metal instrument produced

no sensation when applied to the rectal mucous membrane, but that the anal canal is very sensitive to thermal stimuli. A curious result was obtained by Zimmermann in his recent observations on himself. Although he found that hot and cold water had no effect and a cold glass rod passed 10 cm. through a tube into his rectum produced no sensation, a hot rod produced a pleasant sensation of warmth in the abdomen, which remained unaltered in character even when it was heated to 140° C.! L. R. Müller records a similar observation.

In a paper published at the same time as our first communication on the subject, Becher described experiments which showed that he differed from most people in that he experienced no sensation of any sort after hot and cold water had passed his pharynx. A similar variability in the perception of heat and cold to that which occurs in the œsophagus of different individuals may perhaps also exist in the stomach and intestines and account for the contradictory results obtained by different observers. My own conclusion would be that *the œsophagus and anal canal are almost always sensitive to heat and cold, the stomach rarely, if ever, and the colon to a limited extent in a very small proportion of cases.*

It has often been suggested that discomfort and pain in the stomach in the absence of organic disease may be due to hyperæsthesia of the mucous membrane, which becomes capable of producing sensations in response to stimuli, which are normally without effect. I had the opportunity of testing this theory in a case, in which it seemed possible that the symptoms might be due to hyperæsthesia of the gastric and intestinal mucous membrane to thermal stimuli. A man, 52 years old, complained of a burning sensation inside his abdomen, which had been present for thirty years, but had recently become almost unbearable. The sensation was constantly present and was worse in hot weather. Hot food produced an immediate aggravation, and the patient was convinced that he could feel it enter his stomach, which was the chief but not the only seat of the burning sensation. For years he had taken nothing but cold food and had been in the habit of drinking iced water at all hours of the

day. This always produced a temporary diminution in the sensation of heat, and he obtained a similar result by injecting ice-cold water into his colon. After blindfolding him, we poured very hot and very cold water into his stomach and colon through a double tube : he did not feel the slightest thermal or other sensation. The feeling of heat must therefore have been entirely mental in origin, and the relief given by cold drinks and cold enemata the result of suggestion.

[1] H. Quincke : *Arch. f. exp. Path. u. Pharm.*, xxv, 375, 1889.

[2] A. Neumann : *Wien. klin. Woch.*, xix, 923, 1906.

[3] J. Ch. Roux : *Maladies du Tube Digestif*, edited by Debove, Achard and Castaigne, i, 375. Paris, 1907.

[4] L. R. Müller : *Mitteil. aus den Grenzgeb. d. Med. u. Chir.*, xviii, 600, 1908.

[5] E. Becher : *Zeitschrift f. Psychologie*, xlix, 341, 1908.

[6] E. H. Weber in R. Wagner's *Handwörterbuch d. Physiologie*, Bd. iii, 2. Abtheil., 497. Braunsweig, 1846.

[7] H. Head, W. H. Rivers, and J. Sherren : *Brain*, xxviii, 99, 1905.

[8] *Guy's Hospital Reports*, lxi, 401, 1907.

[9] James Mackenzie : *Symptoms and their Interpretation*, p. 125. London, 1909.

[10] A. F. Hertz, F. Cook, E. G. Schlesinger : *Journ. of Physiology*, xxvii, 481, 1908.

[11] R. Zimmermann : *Mitteil. aus den Grenzgeb. d. Med. u. Chir.*, xx, 445, 1909.

[12] Steinhäuser: *Experimenta nonnulla de sensibilitate et functione intestini crassi*, p. 19. Lipsiæ, 1831. Quoted by E. Meumann, *Arch. f. d. ges. Psychologie*, ix, 26, 1907.

CHAPTER III

SENSATIONS PRODUCED BY CHEMICAL STIMULATION

(*a*) **Hydrochloric and organic acids.**—The observations [1], which have hitherto been published on the sensibility of the gastric mucous membrane to hydrochloric acid, are so contradictory that no reliable conclusions can be drawn from them.

Our own observations show that 0·4 and 0·5 per cent. hydrochloric acid, introduced into the œsophagus or the empty stomach of normal individuals through a tube, produces no sensation of any kind. The introduction of 0·5 per cent. hydrochloric acid through the gastrostomy wound into the stomach of patients with œsophageal stenosis also produces no sensation.

Since the preliminary account of our observations was published, they have been confirmed by J. E. Schmidt,[2] who found that the introduction of from 15 to 30 c.c. of 0·5 to 2·0 per cent. hydrochloric acid through a gastrostomy wound into the empty stomach of three patients and through a stomach-tube into his own stomach produced no sensation.

It is thus clear that the pain, which is often associated with the presence of excess of free hydrochloric acid in the stomach, cannot be due, as has generally been believed, to the excess of hydrochloric acid alone, as it is certain that as much as 0·5 per cent. free hydrochloric acid is never present in the stomach. Moreover many cases in which excess of free and of active hydrochloric acid is discovered by chemical analysis are not associated with pain or other gastric symptoms, and the excess present in painful conditions may persist after the pain has been completely relieved by treatment. We may conclude that the presence of an excessive quantity of hydrochloric acid in the stomach

is only one manifestation of a disease, in which pain is another, and that it is not the cause of the pain.

As 0·5 per cent. hydrochloric acid produces a smarting pain when applied to an abrasion of the skin, it might be expected that it would cause pain when applied to a gastric ulcer. But in six cases, in all of which the diagnosis was confirmed by operation, four ounces of 0·5 per cent. hydrochloric acid, introduced by a tube into the empty stomach, produced no sensation whatever. The same result was obtained in another case, when a mixture of hydrochloric acid with pepsin was used. These observations show that, contrary to the common belief, contact with free hydrochloric acid is not the direct cause of the pain in gastric and duodenal ulcer. That the acid is, however, in some way connected with the production of the pain there can be no doubt, as rapid relief always follows the administration of an alkali. This question will be discussed in detail in a later chapter.

The sensation of heartburn or pyrosis has often been ascribed to the regurgitation of hydrochloric acid into the œsophagus. Our observations, however, negative this theory.

We have found that 1 per cent. lactic acid, 1 per cent. butyric acid and 1 per cent. acetic acid produce no sensation when introduced into the œsophagus or stomach. As the quantity present in the severest cases of gastric fermentation is never as great as this, these acids can also have no direct relation to the production of pain or heartburn.

(*b*) **Alcohol and carminatives.**—Our first experiments were done with *Crême de menthe*, and in order to separate the effects of the peppermint from those of the alcohol, we also used peppermint water and a solution containing 48 per cent. alcohol with 28 per cent. cane-sugar, these amounts being the same as those present in the liqueur. *Crême de menthe*, introduced by a tube into the empty stomach, gives rise to a sensation of warmth which may occur immediately, but more frequently only develops in the course of a minute or two. A similar result was obtained with the 48 per cent. alcohol, but not with the peppermint

water, so that it is the alcohol and not the peppermint, which produces the sensation after the liqueur is introduced into the stomach. *Crême de menthe* also produces an immediate burning sensation in the epigastrium when poured through a tube into the lower end of the œsophagus. This is a much stronger sensation than that produced in the stomach, but it is impossible to distinguish the locality of the two. The burning sensation in the œsophagus is produced by the alcohol, as the 48 per cent. solution has the same effect, whilst the peppermint water, if warmed to the body temperature, produces no sensation. It may therefore be concluded that the immediate burning sensation produced by a liqueur is in the œsophagus, and that the subsequent feeling of warmth is in the stomach. Both are due to the alcohol alone. It was found that the lower end of the œsophagus is so sensitive to alcohol that special precautions had to be taken in testing the sensibility of the stomach in order to prevent a few drops reaching the œsophagus when the tube was withdrawn. The tube was kept in the stomach for half a minute after all the fluid had been poured down, and any remaining drops were washed away by a little water before it was removed.

We have confirmed our results by observations on patients, on whom a gastrostomy had been performed for epithelioma of the œsophagus. On pouring a 48 per cent. solution of alcohol by a tube into the artificial opening of the stomach, a sensation of warmth was produced either immediately or after a short latent period. One patient, who had felt nothing when hot water had been introduced, although he was blindfolded and had no idea what was being done, spontaneously stated that the sensation was more like that produced by swallowing spirits than by drinking hot water, and that it could be better described as ' burning ' than merely warm.

In another man, on whom a gastrostomy had been performed, we found that the introduction of two ounces of 5 per cent. alcohol produced a definite sensation of warmth after a latent period of half a minute ; the sensation gradually faded away after three minutes. In a normal individual

25 per cent. alcohol produced a burning sensation, but five ounces of 1 per cent. alcohol had no effect.

In the patient with bulbar paralysis already referred to, Dr. Johnson and I found that the sensation of warmth in the œsophagus and stomach produced by alcohol was unaffected, in spite of the impairment in the sensibility to other stimuli which had resulted from the disease of the vagal nuclei. It is possible therefore that the sensation of warmth, which is produced by alcohol and which is undoubtedly different in character to that produced by hot fluids, is conveyed by sympathetic nerves and not by the vagi.

Alcohol in a strength greater than 25 per cent. gives rise to a sensation of burning when applied to the mucous membrane of the anal canal, but when introduced into the pelvic colon through a sigmoidoscope or into the more proximal parts through a colostomy opening, a strength of 50 or even 90 per cent. is required. After a sensation of heat has been produced, the repetition of the experiment a few minutes later may have no effect owing to the protection of the mucous membrane by mucus, which is secreted as a result of the irritation caused by the alcohol.

Heartburn is the hot sensation felt in the epigastrium in many cases of dyspepsia. It is frequently accompanied by regurgitation of fluid, which produces a similar sensation beneath the sternum and a sensation of scalding in the pharynx. We have shown that the presence in the stomach or œsophagus of excess of free hydrochloric acid and of the organic acids, which result from fermentation in the stomach, cannot be the cause of these symptoms. The burning sensation produced by alcohol in the stomach closely resembles heartburn, and the stronger sensation it produces in the œsophagus resembles the substernal burning sensation which may accompany heartburn. It therefore seems possible that the alcohols, which may be formed in the stomach in considerable quantity by the action of yeast and sarcinæ on carbohydrates, are the cause of heartburn when present in excess in the stomach, and that their regurgitation into the œsophagus gives rise to the burning sensation which may

be felt beneath the sternum in such cases. The scalding in the pharynx is probably due partly to alcohol, but more to the organic acids with which it is commonly associated.

(*c*) **Glycerine.**—It has generally been supposed that the sensation of burning and tenesmus produced by a glycerine suppository or enema is due to its action on the rectal mucous membrane. With the aid of a rectal speculum Dr. Johnson and I found, however, that glycerine produces no sensation when applied to the rectal mucous membrane, and that the characteristic sensation only occurs when it is applied to the anal canal. The sensation produced by a glycerine enema or suppository must therefore be due to a small quantity escaping from the rectum into the anal canal, and the defæcation reflex to which it gives rise probably originates in the anal canal and not as formerly believed in the rectum.

In one individual examined with Mr. Barber glycerine produced no sensation in the anal canal. We found subsequently that a glycerine suppository produced no burning or tenesmus or only the very slightest desire to defæcate. Perhaps a similar insensibility of the anal mucous membrane accounts for the inefficacy of glycerine suppositories and enemata, which is occasionally observed in patients with retention of fæces in the rectum.

We may conclude that *the mucous membrane of the œsophagus and stomach, whether intact or ulcerated, is insensitive to stimulation by dilute hydrochloric acid and dilute organic acids, that the rectum is insensitive and the anal canal is sensitive to stimulation by glycerine, and that the contact of alcohol with the mucous membrane of all parts of the alimentary canal gives rise to a sensation of heat.*

[1] S. Talma: *Zeitschrift f. klin. Med.*, vii, 407, 1884; M. Löwenthal: *Berl. klin. Woch.*, xxix, 1188, 1892; M. Bönniger: *Berl. klin. Woch.*, xlv, 396, 1908; D. Heineke and M. van Schus: *Arch. des Mal. de l'Appar. dig.*, ii, 467, 1908.

[2] J. E. Schmidt: *Mitteil. aus den Grenzgeb. d. Med. u. Chir.*, xix, 278, 1909.

CHAPTER IV

THE SENSATION OF FULNESS AND DISTENSION

The study of the effect of tension on the muscular coats of the hollow viscera is of great importance, as I believe that a large proportion of abnormal abdominal sensations depends upon the stimulation of the sensory nerves of involuntary muscle-fibres. Some suspicion of this may fifty years ago have prompted Brinton[1] to write that 'the indistinct sensation of the healthy stomach affords us the best clue to the acute sensibility of the diseased one; and allows us to trace a scale—from satiety to repletion; from repletion to distension and weight in the epigastrium; and from hence to the dull, heavy aching of dyspepsia, the gnawing or burning pain of ulcer, and the sharp agony of cancer of the stomach'.

(*a*) **The sensation of fulness in the œsophagus.**—With Dr. Hill and Mr. Harrison, and with Mr. Barber, I have investigated the sensation produced by distension of the œsophagus. A thin india-rubber bag attached to a gum-elastic catheter was passed various distances down the œsophagus in two normal men after the pharynx had been rendered anæsthetic by means of cocaine. When the slight initial discomfort caused by the introduction of the instrument had disappeared, we inflated the balloon by means of a valved pump, the tension exerted on the œsophagus by the distended balloon being measured by a manometer connected with the apparatus. On slowly inflating the balloon, a sensation of fulness situated deeply beneath the sternum in the middle line was felt as soon as the pressure reached 44 and 54 mm. of mercury respectively in the two cases. The sensation did not diminish even when the tension was maintained for as long as fifteen minutes, and there was no fall in pressure, such as would be produced if relaxation

of the tone of the œsophagus occurred. The sensation disappeared in three seconds when the air in the balloon was suddenly allowed to escape. When the balloon was slowly inflated to a greater degree so as to exert a higher tension, the sensation of fulness was increased, but its character remained unaltered. In each case a feeling was experienced, which suggested that a lump had become lodged in the œsophagus. This seemed to be larger and the sensation was more unpleasant, the greater the intra-œsophageal pressure. The sensation of fulness felt as if it were produced in the middle line deeply beneath the anterior surface of the body ; it was never felt posteriorly. Its level was accurately recognized, the error never exceeding one inch.

At intervals, varying in duration from ten to sixty seconds, whilst the balloon remained inflated, a rise in pressure of between 30 and 40 mm. was recorded. This was often associated with an increase in the local sensation of fulness, and occasionally a sensation of uncomfortable fulness was also felt behind the cricoid cartilage, from which it passed rapidly down to the position of the balloon, where it reached its greatest intensity ; after a few seconds it disappeared without passing any further down. The rise in pressure and the accompanying sensation appeared to be due to a peristaltic wave. No rise of pressure and no change in the sensation followed a voluntary act of deglutition, which does not therefore necessarily result in a peristaltic wave. It seems clear that the presence of a foreign body within the œsophagus gives rise to peristaltic waves originating at its upper end and passing down as far as the foreign body, the waves having the object of dislodging it. Similar peristaltic waves occurred when the balloon was not distended and its presence was unfelt ; the pressure never reached the minimal pressure required to produce a sensation of fulness, and consequently their passage was not felt.

The sensation of fulness produced by distension of the œsophagus is probably due to tension on the fibres of its circular muscle coat ; it is in fact a form of muscle-sense. The sensation is probably carried by the vagal nerves, as in our patient with bulbar paralysis Dr. Johnson and I found

that no sensation of fulness was felt until the tension exerted by the balloon on the œsophageal wall reached 90 mm. of mercury, which is approximately double the normal minimal stimulus.

The sensation of fulness in the œsophagus may be felt under natural conditions. Our X-ray observations showed that a large bolus of food often passes very slowly down the œsophagus and may even remain in one position for several minutes. This is apt to occur particularly at the level of the bifurcation of the trachea, as it is at this point that the œsophagus is most narrow. The feeling of pressure behind the sternum, which is sometimes experienced when food is insufficiently chewed, may therefore be due to a solid lump of food becoming lodged in one position for a considerable time, after which it moves slowly downwards. A similar less prolonged sensation is produced when a single large and hard mouthful of food is swallowed; the common belief that the food has 'stuck' in the œsophagus under these circumstances is probably a correct interpretation of the sensation. The sensation disappears after drinking fluid, corresponding with the fact that drinking can be seen with the X-rays to dislodge with more or less difficulty a bolus sticking in the œsophagus.

A similar sensation is produced in cases of organic œsophageal obstruction after food has been swallowed, doubtless owing to the distension of the œsophagus immediately above the obstruction. In slight cases it gradually disappears as the food passes on and the distension diminishes, whilst in severer cases relief may only occur after the food has been rejected through the mouth.

The power of localization of the muscle-sense in the œsophagus appears to be as accurate under these natural conditions as it was in our experimental investigations. I have several times observed that when a patient with œsophageal obstruction is given a bismuth meal and is asked to point to the place where the passage of the food seems to be obstructed, the position indicated is within an inch of the level of the obstruction. Similar accuracy of localization was reached in four-fifths of the 134 cases of cancer

of the œsophagus studied by Lamy [2] in Mathieu's clinique. In the remaining fifth, however, the sensation of obstruction or fulness was felt in the upper third of the œsophagus, although the stenosis was really in the lower third. This was doubtless due, as Lamy suggested, to obstruction by a reflex spasm in the cervical region. Thus Dr. Hill and Mr. Harrison tell me that they have found in cases of cancer situated in the lower third of the œsophagus, in which a sensation of obstruction was felt high up, that a spasmodic contraction was present near the upper extremity, which made it difficult to pass an œsophagoscope without a general anæsthetic.

The sensation of obstruction always gives the impression of being deeply situated in the middle line beneath the trachea, the sternum, or—in the case of obstruction near the cardiac orifice of the stomach—in the epigastrium. One patient, however, with a growth of the cardia felt the sensation equally in the epigastrium and at the same level behind.

(*b*) **The sensation of fulness in the stomach.**—When an individual is hungry and food is eaten, the sensation of emptiness in the upper part of the abdomen gradually disappears, and if the meal is large enough it is gradually succeeded by a feeling of fulness. The more rapidly a meal is eaten, the sooner is the sensation of fulness experienced; it is more likely to follow a small meal which has been bolted than a large meal which has been eaten slowly.

With the object of discovering the origin of the sensation of fulness in the stomach, Dr. Johnson, Mr. Barber and I have investigated its sensory reaction to alterations in intragastric pressure by inflating the stomach of two healthy men with air through a tube connected with a manometer. We found that a sensation of fulness or tightness in the upper part of the abdomen, associated with a desire to eructate, was felt as soon as the intragastric pressure reached respectively 12 and 14 mm. of mercury in the two cases. The pressure fell after twenty seconds by approximately 2 mm., owing apparently to relaxation of the tone of the stomach, and simultaneously the sensation of fulness dis-

appeared. On now slowly injecting more air, the pressure gradually rose to its original height and the sensation reappeared; it again disappeared after twenty seconds, the pressure simultaneously falling 2 mm., after which it remained constant. Exactly the same rise in pressure and the same sensation of fulness, followed by a fall in pressure and the disappearance of sensation, were produced four times in succession by injecting air, none being allowed to escape in the interval.

The sensation was felt deeply beneath the upper part of the anterior abdominal wall in the middle line, nothing being felt behind. Percussion showed that the upper and lower limits of the distended stomach did not accurately correspond with the limits of the sensation.

These observations prove that the tension exerted from within on the circular muscle-fibres of the stomach is the cause of the sensation of fulness. Owing to the great variations in the tone of these muscle-fibres under different conditions, the volume of contents necessary to produce the tension, which will act as an adequate stimulus to the muscle-sense, varies considerably. In normal individuals, as Moritz[3] was the first to show, the tone can undergo such great variations that the intragastric tension remains between 4 and 6 mm. of mercury whether 50 or 500 c.c. of food are introduced, so long as the introduction is sufficiently slow to allow the reflex relaxation of tone to occur. Grützner[4] has, however, pointed out that a mere relaxation of the tonic contraction of the fibres of the muscular coat of the stomach is quite insufficient to account for the enormous variations, which may occur in the volume of its contents without any accompanying change in the internal pressure. Thus Albert Müller[5] found that the length of the muscle-fibres of the filled stomach of a frog was only one and a half to three times that of the fibres in the empty and contracted stomach, although the circumference of the former was five times that of the latter. Grützner had suggested that a re-arrangement of the muscle-fibres must occur, and Müller found that this was indeed the case, the fibres being arranged in from fifteen to twenty layers in the

contracted stomach and in only two or three layers in the full stomach.

It follows from the work of Grützner and Müller that the muscle-fibres of the stomach are completely relaxed some time before its greatest capacity is attained. Müller and Saxl [6] suggested that the re-arrangement of the individual muscle-fibres, like the relaxation of their tone, is due to a reflex from the œsophagus during swallowing. It is difficult to see by what nervous mechanism such a re-arrangement could occur, and it seems to me much more probable that it only occurs in response to an increase in intragastric tension, and that in man this tension is sufficient to produce the sensation of fulness. When, therefore, an excessive quantity of food is eaten, the sense of fulness is experienced as soon as muscular relaxation is complete, as the increase in intragastric tension, which is necessary for the production of a further increase in capacity, then takes place.

In normal individuals the sensation of fulness can also be produced by eating very rapidly, as under these circumstances insufficient time is allowed for the reflex relaxation of the tonic contraction of the muscle-fibres, and consequently the intragastric pressure rises and the adequate stimulus is produced.

Under pathological conditions the tone of the stomach may be permanently increased and complete relaxation may be impossible. A comparatively small quantity of food will then cause an uncomfortable sensation of fulness, which can only be prevented by eating very slowly. In a patient recently seen, the walls of the stomach were so infiltrated with growth that they could not relax ; the capacity of the stomach was thus extremely small and could not be increased by relaxation of the muscular coat when the volume of its contents increased. Consequently a very small quantity of food sufficed to produce a rise in intragastric pressure, and the patient complained that almost immediately after he began a meal he felt as full and distended as if he had eaten an enormous quantity.

The sense of fulness, which is a common symptom in patients with atonic dilatation of the stomach, however

slowly food is eaten, is due to the muscle-fibres being already completely relaxed before any food is taken ; the increased dilatation, which has been shown by the X-rays to occur as each additional quantity of food enters the stomach, must be due to re-arrangement of the muscle-fibres, and, as this is only induced by an increase in their tension, a sensation of fulness results. In slighter cases the muscle-fibres are not completely relaxed, but complete relaxation occurs as soon as a small quantity of food has been eaten. Any further addition to the bulk of gastric contents will then give rise to a feeling of fulness.

A sensation of fulness in the stomach may thus result from two diametrically different conditions—excessive and deficient gastric tone. The impossibility of distinguishing between these conditions from the nature of the sensation is well illustrated by the fact that among a number of medical men, who have consulted me for what they believed was atonic dilatation of the stomach on account of the sense of fulness they experienced after eating a comparatively small quantity of food, nearly as many were found to have small hypertonic stomachs as large atonic stomachs.

Just as it is impossible to distinguish between the sensation of fulness resulting from tension on tonically contracted and on completely relaxed muscle-fibres, so is it impossible for a patient to recognize from his symptoms whether the tension which causes the sensation is produced by food or flatulence. I have seen a large number of patients, in whom an X-ray examination in the vertical position showed that no excess of gas was present, although they complained of a sense of fulness in the stomach, which they believed was due to flatulence. As a matter of fact it is very exceptional for the production of gases in the stomach by fermentation to be sufficiently rapid to produce any increase in intragastric tension, for relaxation occurs, just as it does when food is taken slowly into the stomach. When, however, air is swallowed in excess, as occurs in many patients with nervous dyspepsia and in some with organic diseases such as gastric and duodenal ulcer, the quantity of gas in the stomach increases so rapidly that a corresponding

relaxation cannot take place and a rise in tension results. As it is impossible for a patient to distinguish between a feeling of fulness due to tension produced by food and tension produced by flatulence, he generally assumes that it is a result of the latter and imagines that he can 'disperse' it by means of eructation. This is probably the result of an experience, perhaps many years before, in which an attack of acute indigestion, associated with the production of an excessive quantity of gas by fermentation, led to a sense of fulness, which, being really due to gas, was relieved by eructation. Thus some patients who complain of a sense of fulness, which is apparently identical with that felt by the majority of patients who complain of what they believe is flatulence, do not associate the sensation with flatulence and make no effort to eructate, probably because they have never had the experience of an attack of indigestion with fermentation, in which relief was at once obtained by eructation. When the sense of fulness is not due to gas, the attempt to eructate is naturally unsuccessful, but the attempt may result in the swallowing of air. Often half a dozen or more attempts are made without success, air being swallowed at each attempt. The feeling of distension consequently increases, until finally the stomach becomes so distended with gas that an attempt to eructate is at last successful, and the feeling of fulness, which is at the end largely due to the increased intragastric pressure brought about by the rapid distension of the stomach with swallowed air, is relieved.

Kelling [7] has demonstrated that a reflex mechanism exists, by means of which the tone of the muscles of the abdominal wall adapts itself to the varying volume of the contents of the alimentary canal. In the absence of such a mechanism, the consumption of a meal of moderate size would result in a considerable rise of intra-abdominal pressure and consequently of intragastric pressure. Thus I have found that the volume of the abdominal contents varies between 4,000 and 5,000 c.c. ; after a moderately large meal with a bulk of 1,000 to 1,500 c.c., the volume of the abdominal contents must therefore increase by 25 to 35 per cent..

When a meal is rapidly eaten, it might be expected that there would be insufficient time for the proper relaxation of the abdominal wall as well as of the stomach, and that consequently the general intra-abdominal tension as well as the intragastric tension would be increased. The sense of fulness which follows might therefore be a result of tension on the abdominal muscles as well as on the wall of the stomach, and Becher has even suggested that the tension on the various structures of the abdominal wall is the sole cause of the sensation. Such a view, however, cannot be accepted, as the sensation may be felt by an individual whose abdominal wall is so lax that it can never be stretched when he is lying down, however full the stomach may be. Moreover, the exactly analogous sensation produced in the œsophagus and rectum can only be visceral in origin, and our experiments, which I shall describe later, on the sensation of fulness in the intestine prove that this also is independent of the abdominal muscles. A further proof in the case of the stomach is afforded by the result of inflating the stomach by the carbon-dioxide given off when solutions of six grammes each of sodium bicarbonate and tartaric acid are separately drunk. Under atmospheric pressure at the body temperature 1,700 c.c. of carbon-dioxide are produced. Kelling [8] showed that in normal individuals the tone of the stomach is such that sudden inflation with from 600 to 1,600 c.c. of gas produces a sufficient rise in intragastric pressure to act as an adequate sensory stimulus. The pressure produced by the sudden formation of 1,700 c.c. is thus so great that normal individuals invariably complain of an unpleasant and often painful sense of fulness, which is relieved by eructation. On the other hand, in patients with atonic dilatation the tone of the muscular coat of the stomach is so feeble that the potential capacity of the stomach, even when empty, is more than 1,700 c.c., and consequently this volume of gas produces no rise in intragastric pressure. If the sense of fulness depended upon stretching of the abdominal wall, it would follow inflation of an atonic stomach, as the volume occupied by the gas is greater than in the normal stomach owing to the smaller intragastric pressure; but no sensation of fulness

is felt, and the patient may actually feel more comfortable after the inflation. It may therefore be concluded that the sensation of fulness in the stomach is due to tension on its muscular coat, and depends very little and only in extreme cases on stretching of the abdominal wall.

(*c*) **The sensation of fulness in the intestines.**—I have had no opportunity of investigating the sensation of fulness in the small intestine experimentally, but Mr. Digby and I have made numerous observations in the colon by introducing the apparatus I have already mentioned in connexion with the œsophagus through a colostomy opening. The results obtained can probably be directly applied to the small intestine.

As soon as the sensation produced in the abdominal muscles by the introduction of the instrument into the colon had disappeared, we inflated the balloon. When a certain internal pressure was attained, a sensation described by the patient as 'fulness' or 'wind' was felt. In our earliest experiments we did not use a manometer, and we were thus unable to estimate the pressure which was being exerted on the intestines; in two patients inflation produced no sensation, but this was doubtless due to the pressure being insufficient, as we did not like to inflate the bowel to any considerable degree without knowing how great a pressure we were exerting. These experiments suggest that the sensation of fulness in the intestines is due, as in the case of the stomach, to tension on the muscular coat.

Three of our patients tried to eructate during the experiment with the object of getting rid of the flatulence which they thought was present. This shows how difficult it is to recognize the source of the sensation. It is always felt at a lower level in the abdomen than that due to gastric distension, but its character is so similar that a patient, who is aware of the relief sometimes afforded by eructation to the sensation of fulness in the stomach, hopes to obtain by this means the same relief, although the sensation is produced by distension of his colon.

Before concluding that the sensation of fulness felt in our experiments was produced in the viscera themselves,

it was necessary to exclude the possibility that it resulted from stretching of the peritoneal attachment of the bowels or from pressure on the anterior abdominal wall. This was done by observations on a patient with a prolapse of two and a half inches of his pelvic colon through the gap left by the removal of part of the sacrum in the performance of Kraske's operation for excision of the rectum. Although pinching produced no sensation, a sensation of ' wind ' was felt below the umbilicus when the exposed intestine, the attachment of which was kept relaxed, was inflated by a balloon.

In several experiments on other patients a finger was introduced through a colostomy opening ; after the initial discomfort caused by the stretching of the gap in the abdominal wall had subsided, the tip of the finger was bent so as to press through the intestine upon the anterior abdominal wall sufficiently to produce a distinct prominence. In the majority of cases no sensation was produced, thus proving that mere pressure from within on the structures of the anterior abdominal wall is not the cause of the sensation of fulness in the abdomen. Even squeezing the abdominal wall between one finger inside and one outside produced nothing more than a local sensation in the muscle and skin. In two cases, however, a sensation of fulness and pain was produced by slight and strong pressure respectively ; this was quite exceptional and was probably due to the tension exerted on the muscular coat of the bowel, as the descending colon, which was the part of the bowel involved, is fixed posteriorly.

The muscular coat of the intestines undergoes variations in tone as great as those of the muscular coat of the stomach. This is well seen in the iliac colon, which, when empty, can often be felt as a contracted cord, indicating that its lumen is almost completely obliterated. Some hours later, when fæces and gas are traversing it on their way to the pelvic colon, its diameter may be 3 cm. or more. The tension exerted by the intestinal contents on the muscular coat is under normal conditions the same whether the bowel be empty and contracted or full and dilated, as the passage of fæces and gas is sufficiently slow to allow the tone to diminish *pari passu* with the increase in their volume. Distension is

the most important stimulus of peristalsis, but the adequate tension for the production of peristalsis is lower than that for the production of sensation, as peristalsis is constantly produced in this way and is under natural conditions never associated with any sensory manifestation. When, however, excessive bacterial decomposition occurs in the intestine, the production of gas may be so rapid that the relaxation of the intestinal tone is unable to keep pace with it, and the intracolonic pressure rises sufficiently to produce a sensation of fulness. This rarely amounts to more than a vague sensation of abdominal discomfort so long as the lumen of the bowel remains unobstructed, as the gas can pass onwards without difficulty to be expelled as flatus *per anum*. If, however, the expulsion of gas is voluntarily restrained, the further rise of tension results in a sensation of fulness, which is instantaneously relieved when flatus is allowed to escape. A more common cause of the sensation of fulness is obstruction to the lumen of the bowel. In constipation temporary obstruction by a hard mass of fæces, upon which the colon has firmly contracted, often occurs; although bacterial decomposition is generally diminished rather than increased in constipation, the gas normally present in the bowel may cause distension, as its onward passage is delayed. In organic obstruction the distension and its sensory results are much more marked, as the production of gas by fermentation and putrefaction is then frequently excessive, and the obstruction prevents its passage into the more distal parts of the bowel.

I have recently seen two patients, upon whom gastroenterostomy had been performed respectively nine and three years previously, in the one case for cicatricial pyloric obstruction and in the other for duodenal ulcer. They were both relieved of their old symptoms, but complained of a disagreeable sensation of fulness immediately above the umbilicus during and directly after every meal, unless they ate very little and very slowly. Both were now supposed to be suffering from atonic dilatation of the stomach. Examination with the X-rays showed, however, that the stomach was small and hypertonic; the stoma was still large

and food could be seen to run through it with such rapidity that the stomach was almost empty a quarter of an hour after half a pint of porridge with two ounces of bismuth oxychloride had been eaten. The rapid distension of the jejunum immediately below the stoma, a part which is never distended under normal conditions, was probably the cause of the sensation of fulness, as its diameter was seen by the X-rays to be unusually great. This explanation is confirmed by the situation of the sensation, which was lower than that due to gastric distension and corresponded with the upper limit of the situation of the pain felt when the small intestine is subjected to rapid distension.

(*d*) **The sensation of fulness in the rectum and the call to defæcation.**—Owing to the tonic contraction of the rectum and the acute angle formed at the pelvi-rectal flexure, the further passage of fæces is obstructed at this point. Consequently, as O'Beirne [9] was the first to show, the pelvic colon becomes filled with fæces from below upwards, and the rectum remains empty until immediately before defæcation. This process of accumulation in the pelvic colon is normally unaccompanied by any sensation, but if an excessive quantity collects owing to obstruction at the pelvi-rectal flexure, discomfort and subsequently pain are felt just above the pubes.

In individuals whose bowels are opened regularly every morning after breakfast, the entry of fæces into the rectum gives rise to the sensation of fulness, which leads to the desire to defæcate and may be termed *the call to defæcation.* The passage of fæces from the pelvic colon into the rectum is the result of active peristalsis in the former, brought about reflexly by various stimuli, the chief of which is the taking of food at breakfast into the empty stomach. The same effect is produced by a cold bath, by the muscular activity involved in getting up and dressing, and by drinking a glass of cold water before breakfast.

The insensibility of the rectum to tactile and chemical stimulation shows that the call to defæcation cannot be due to the mere contact of fæces with the rectal mucous membrane. From analogy with the effect of distension in other

parts of the alimentary canal, it seemed to me that the sensation of fulness in the rectum would probably also be due to its muscle-sense. Observations carried out at different times with Mr. Barber and with Mr. Digby have proved that this view is correct. On introducing an india-rubber balloon connected with a pump and manometer through a sigmoidoscope into the pelvic colon immediately above the pelvi-rectal flexure, slow inflation produced a sensation of fulness in the middle line a little above the pubes, but never in the back. When, however, the rectum immediately below the flexure was inflated, the sensation was no longer felt in front, but in the rectum or in some individuals in the neighbourhood of the sacrum, and when the lower part or ampulla of the rectum was inflated, the sensation was always felt in the rectum itself. Whichever part of the rectum was inflated, the sensation was always accompanied by a desire to defæcate; it was in fact identical with the natural call to defæcation. The minimal pressure required to produce a sensation is greater in the pelvic colon than in the upper part of the rectum, and greater in the latter than in the ampulla; in one individual, for example, the adequate pressures were 110, 85 and 50 mm. of mercury just above the flexure (18 cm. from the anus), just below the flexure (12 cm. from the anus) and in the ampulla of the rectum (8 cm. from the anus) respectively.

Owing to the sudden change in the localization and character of the sensation produced by distension when the pelvi-rectal flexure is passed, the entrance of fæces into the upper part of the rectum at once produces the call to defæcation; this increases in intensity as more fæces enter and the most advanced portion reaches and distends the more sensitive ampulla. If the rectum were the part of the bowel in which the fæces collected, as has frequently but erroneously been stated, the regular return of the desire to defæcate each morning would be impossible, for there would be no sudden entry of fæces, such as is necessary to produce the increase in tension which acts as the adequate stimulus for the call to defæcation.

As the specific sensation, which I have designated the

call to defæcation, is peculiar to the rectum, it is never experienced after excision of the rectum. This was well seen in a patient from whom Mr. R. P. Rowlands had removed the whole rectum from below, the end of the pelvic colon being brought down through the sphincter ani and joined to the skin at the anal margin. The patient stated that he had never felt the ordinary call to defæcation since the operation ; he attempted to open his bowels regularly, as he knew that it was best for him to do so, but he only recognized that there was anything to expel by the occurrence of a feeling of tightness just within the anus, when the first portion began to leave the rectum. Mr. Barber and I found that distension of the bowel immediately above the sphincter caused a sensation of fulness or pain according to the rapidity of inflation ; in both cases it was felt just above the pubes and there was no rectal sensation of any kind. The adequate pressure was approximately the same as in our experiments on inflation of the pelvic colon of normal individuals through a sigmoidoscope. On placing the balloon so that it was grasped by the sphincter ani and then inflating it, the sensation which the patient was accustomed to feel when his bowels began to open was experienced. Exactly the same results were obtained in the case of a woman, from whom Mr. Rowlands had removed six inches of the pelvic colon and the whole rectum by the abdomino-anal method.

The intrarectal pressure, which is adequate to produce the call to defæcation, is constant for each individual. It amounted in different individuals examined under exactly similar conditions to 50, 54, and 58 mm. of mercury. When the inflation was rapid, the first sensation was produced at a slightly lower pressure. Whatever the rate of inflation, the urgency of the call to defæcation increased as the intrarectal pressure rose. As soon as the inflation was discontinued, the pressure began to fall and the desire to defæcate became less urgent and finally disappeared. When the intrarectal pressure was just sufficient to produce a minimal sensation, the latter disappeared in less than half a minute, the pressure having gradually fallen about 4 mm.. After the sensation had disappeared there was no further fall in pressure. On

now pumping more air into the balloon, the pressure rose ; as soon as it reached the height at which the call to defæcation was first experienced, the latter returned, but gradually disappeared as the pressure fell once more. A similar waxing and waning of sensation and intrarectal pressure was observed several times in succession without allowing any air to escape. It is clear that the size of the balloon required to exert an adequate pressure on the rectal wall steadily increased. This could only result from relaxation of the tone of the rectum, an occurrence which is strictly analogous to what has already been described in connexion with the stomach and intestines.

If a response is not at once made to the call to defæcation, the desire to defæcate passes away ; it has generally been supposed that this was due to the fæces being carried back into the pelvic colon by antiperistalsis. The observations of Tuttle [10] had previously convinced me of the error of this view, an error which is at once obvious if digital examination be made at any time after the desire to defæcate has passed away, as fæces are always found in the rectum. Our experiments show that the true explanation is the relaxation of tone, which occurs in the muscular coat of the rectum after it has been subjected to a certain degree of tension for a short period. The call to defæcation only returns after a further quantity of fæces has entered the rectum and produced a rise in intrarectal pressure. This may occur after any meal, but most frequently only after breakfast on the following morning.

Further observations proved that the minimal pressure required to produce the call to defæcation diminished as the length of the balloon increased. Thus in one individual the pressure was 48, 38 and 32 mm. of mercury respectively with balloons $4\frac{1}{4}$, $5\frac{1}{2}$ and $7\frac{1}{2}$ cm. long. Thus the urgency of the call to defæcation varies with the length as well as with the diameter of a mass of fæces, and the efficacy of an enema depends upon the length of bowel stimulated as well as upon the degree of its distension. Although a large mass of fæces produces a greater desire to defæcate than a small mass at any given moment, it is quite impossible to estimate the quantity of fæces present in the rectum with any

accuracy from the strength of the sensation. For the tone varies greatly from time to time ; a small mass will on one occasion be sufficient to produce the adequate intrarectal tension, whereas on another occasion a mass double the size may have no effect.

As a result of my investigations with the X-rays on the motor functions of the intestines in health and disease,[11] I concluded that all cases of constipation can be divided into two classes : in the first, which may be called *intestinal constipation*, the passage through the intestines is delayed, whilst defæcation is normal ; in the second class, for which I have adopted the term *dyschezia*, there is no delay in the arrival of the fæces in the pelvic colon, but their final expulsion is not adequately performed.

The most common cause of dyschezia is the habitual disregard of the call to defæcation on account either of ignorance or laziness or of fear of pain in diseases of the anus and the neighbouring organs. I have already described how the sensation of fulness in the rectum passes off owing to relaxation of the tonic contraction of its muscular coat, if the call to defæcation be disregarded. If it is again disregarded after its return on the arrival of more fæces in the rectum, further relaxation occurs. More and more fæces accumulate in the rectum, the muscular coat of which becomes more and more relaxed. As the force required to empty the rectum when over-distended with fæces is much greater than that required to empty it under normal conditions, evacuation is now likely to be incomplete, even if a great effort be made. Consequently fæces are constantly present in the rectum instead of only for a few minutes before defæcation, and the lumen of the rectum is permanently increased owing to the atony of its muscular coat. It has sometimes been recommended that patients, who are constipated as a result of irregularity in their habits, should attempt to open their bowels after breakfast, but should not obey the call to defæcation if felt at other times of the day. Our observations show that this teaching is wrong, and that in addition to the regular morning effort a response should be made to every call, however inconvenient the time.

For the occurrence of a call to defæcation always means that for some reason fæces have just passed from the pelvic colon into the rectum ; the relaxation of tone which follows neglect of the call is undesirable, particularly in patients with dyschezia, in whom a certain degree of atony is already present.

Dyschezia may be due to various other causes, such as weakness of the voluntary muscles of defæcation and the assumption of an unsuitable position during defæcation. But whatever the primary cause, the final result is the same. The incomplete evacuation of the rectum results in the accumulation of fæces and in atonic dilatation.

I believed at first that the absence in dyschezia of any sensation when the rectum contained fæces was due to a blunting of the sensibility of the mucous membrane as a result of the irritation produced by the constant presence of fæces. This view was shown to be erroneous by our observations that the rectal mucous membrane is normally insensitive to tactile stimulation, and that the call to defæcation depends upon the sensibility of the muscular coat of the rectum. The few experiments, which I have at present had the opportunity of making, suggest that the muscle-sense is not impaired in most cases of dyschezia, as the intrarectal pressure required to produce the call to defæcation is not greater than in normal individuals. The dyschezia depends upon the atonic dilatation of the rectum, an abnormally large quantity of fæces being required to exert the normal adequate pressure. In extreme cases a blunting of the muscle-sense may also occur ; in such cases the rapid and considerable distension produced by an enema injected with an ordinary syringe fails to produce the artificial call to defæcation, which in most cases of dyschezia results in a movement of the bowels.

There is, however, an entirely distinct class of dyschezia, which depends upon deficiency of the muscle-sense of the rectum. Congenital deficiency probably causes the dyschezia, which occurs not uncommonly in infants, in whom the slight additional distension produced by the introduction of a finger or a piece of soap into the rectum results in an adequate

stimulus. In the majority of cases the muscle-sense develops as the infant grows older, but congenital deficiency is occasionally the starting-point of dyschezia which lasts through life.

The rectal muscle-sense is completely abolished in diseases of the spinal cord, in which the defæcation centre itself or the fibres connecting it with the brain are involved. I have found by means of the X-rays that the severe constipation which occurs in these cases is due to dyschezia, as the rate of passage through the colon is unaffected.

The nerve-fibres, which convey afferent impulses from the intestinal muscles, appear to ascend the posterior columns of the spinal cord along with those from the voluntary muscles, as observations made with Mr. Barber on fourteen consecutive cases show that the degeneration of the posterior columns which occurs in tabes always results in some alteration in the muscle-sense of the rectum. Thus in five cases, in which constipation was a constant and marked symptom, and in one, in which constipation was only occasionally present, the pressure required to produce the call to defæcation was double or more than double that required in normal individuals examined at the same time with the same apparatus. In seven out of the remaining eight cases the adequate pressure was more or less increased; in one who complained of morning diarrhœa, it was unusually low. In all of the fourteen cases the sensation lasted for a much longer period after the pressure was relaxed or the balloon removed than in normal individuals: the duration varied between fifteen seconds and six minutes, whereas normally the sensation always disappears under similar conditions within five seconds. The duration was longest in the patients who complained of morning diarrhœa. One of these opened his bowels six or seven times every morning within a period of an hour and a half, a small solid stool being passed each time; he had, however, found by experience that he could terminate the annoying desire to defæcate by ignoring it and going out of doors, when it gradually disappeared. This so-called morning diarrhœa, which is not uncommon in tabes, is thus probably due to the excessive duration of the sensation of fulness felt

in the rectum after the stimulus which produced it has ceased to operate. Finally, in five out of the fourteen cases the call to defæcation only occurred after a latent period of a few seconds instead of instantaneously as in normal individuals.

We may conclude that *the ill-defined sensations, which occur in all parts of the alimentary canal and which may be grouped together as 'the sensation of fulness', are due to stretching of its muscular coat and constitute a form of muscle-sense*, which is probably shared by all hollow viscera. For each part of the alimentary canal a certain tension, which varies in different individuals, is required in order to produce the first trace of sensation. As the tone of the muscular coat, except in the case of the œsophagus, varies greatly under different circumstances, the volume of contents necessary to produce the adequate tension is as variable as the tension is constant. The relative sensibility of different parts of the alimentary canal to distension cannot be ascertained with any degree of accuracy, as the minimal pressure is greater the smaller the area subjected to the tension, and it is experimentally impracticable to apply pressure to an equal area of the wall of the œsophagus, stomach and intestine. Thus the comparatively low pressure required to produce a sense of fulness in the stomach is doubtless due to the fact that the whole of the stomach was inflated and not merely a small segment as in the case of the œsophagus and intestine. Similarly the pressure required to produce a minimal sensation in the colon was slightly less when a large area was inflated through an open rectal tube than when a limited area was distended by means of a balloon. It may, however, be said that the œsophagus is more sensitive than any part of the intestine except the rectum, that the upper part of the rectum is more sensitive than the pelvic colon, and that the sensibility of the rectum increases as the anus is approached.

It may further be concluded that *the call to defæcation is a specialized form of visceral muscle-sense.* It is the result of the pressure exerted on the rectal wall by the fæces

on their entry from the pelvic colon, the urgency of the call depending upon the tone of the circular muscle-fibres of the rectum at the time and the bulk and consistence of the fæces.

[1] W. Brinton: *Diseases of the Stomach*, 2nd edition, p. 44. London, 1864.
[2] L. Lamy: *Arch. des Mal. de l'Appar. Dig.*, iv, 451, 1910.
[3] F. Moritz: *Zeitschrift f. Biol.*, xxxii, 313, 1895.
[4] P. Grützner: *Ergebnisse der Physiol.*, iii, 2. Abtheil., 73, 1904.
[5] A. Müller: *Arch. f. d. ges. Physiol.*, cxvi, 252, 1907.
[6] A. Müller and P. Saxl: *Wien. klin. Woch.*, xxi, 483, 1908.
[7] G. Kelling: *Zeitschrift f. Biol.*, xliv, 161, 1903.
[8] G. Kelling: *Deutsche med. Woch.*, xviii, 1191, 1892.
[9] James O'Beirne: *New Views of the Process of Defecation.* Dublin, 1833.
[10] J. P. Tuttle: *Diseases of the Anus and Pelvic Colon*, p. 523. London, 1903.
[11] A. F. Hertz: *Constipation and Allied Intestinal Disorders.* London, 1909.

CHAPTER V

THE SENSATION OF EMPTINESS AND HUNGER

In 1836 Tiedemann [1] published an admirable analysis of the sensation of hunger; with the exception of a thesis published in 1892 by Nicolai,[2] I have been unable to find any material contribution to the subject in the last seventy-five years. Tiedemann pointed out that hunger consists of two distinct sensations—the local sensation of emptiness in the stomach and the general sensation of malaise and weakness in the body as a whole. He believed that the former is due to an increased irritability of the sensory nerves of the stomach, arising in a similar manner to the irritability of the nerves of special sense, which occurs when they have remained unstimulated for a certain period. On the other hand, he ascribed the general sensation to the effect of starvation on the blood, the altered condition of which reacts on the tissues of the whole body and especially on those of the nervous system.

The local sensation of hunger only occurs in man when the stomach is empty. Nicolai showed, however, that emptiness cannot be the only factor in its production, as it is often not felt until several hours after every trace of a meal has passed through the pylorus. He found, moreover, that if the stomach was washed out before it was quite empty, hunger was only experienced between two and three hours later.

The gastric constituent of the sensation of hunger can, I believe, be explained most satisfactorily in the light of observations made on dogs by Boldirev in Pavlov's [3] laboratory. He found that periods of motor activity, in which the movements are more energetic than those which take place during digestion, occur at intervals in the stomach and intestines of fasting animals. That the production of hunger

should be in some way connected with these spontaneous movements is suggested by the frequent production of borborygmi, which indicate the occurrence of peristalsis, when hunger is experienced. Under ordinary conditions the gastric and intestinal movements are unfelt. But the production of some slight sensation could be explained, if we accepted Tiedemann's theory that the local sensation of hunger depends upon an increased excitability of the sensory nerves of the stomach, analogous to the hyperexcitability of the special senses after prolonged rest. Tiedemann's theory, as originally propounded by him, is insufficient, as the special senses only show their hyperexcitability when they receive their natural stimulus, whereas the sensation of emptiness is present before the sensory fibres of the stomach are stimulated by the consumption of food, the first effect of the latter being to cause the sensation to disappear. As during hunger the tone and spontaneous movements are increased, the latter would probably give rise to sensation, if the afferent nerve-fibres of the muscular coat of the stomach were in a condition of hyperexcitability.

The sensation of emptiness commonly reaches its maximum immediately before a meal. Corresponding with this Glücksmann [4] has observed in man that the sounds heard in the early morning on auscultation over the pylorus become more frequent when the gastric movements are stimulated by the psychical effect of the sight and smell of breakfast, especially if the meal has been somewhat delayed.

Humboldt tells how in times of flood, when food was scarce, the Otomacs living on the banks of the Orinoco ate daily about a pound of earth, which was moulded into large balls and then baked, in order to satisfy the pangs of hunger. It is also related that earth has been consumed on various occasions in war time when food was unobtainable. The relief experienced can be satisfactorily explained by our theory, as the entrance of indigestible material into the stomach would result in the cessation of the movements, and the distension of the stomach would cause its hyperexcitability to disappear. The temporary relief of hunger produced by drinking water can be similarly explained.

These facts are of practical importance in the dietetic treatment of obesity. Big eaters look for a sense of fulness after meals and are unsatisfied by a smaller volume of food than that habitually consumed, even if it has an equal or greater energy value ; conversely they are satisfied with food of smaller energy value so long as its bulk is unaltered. Consequently bulky non-nutritious food should form an important part of the diet of the obese.

The feeling of emptiness in the stomach is much more marked six hours after breakfast than after a fast of twelve hours, which include the night's rest, although the stomach in the latter case has been empty for a longer period. This is probably due to the fact that muscular activity increases the tendency to spontaneous movements of the empty stomach. Thus it is not an infrequent occurrence for hunger, which is absent on waking in the morning, to be felt during and immediately after a cold bath, and still more strongly after a brief period of exercise taken before breakfast.

In all records of voluntary starvation for long periods, hunger is described as increasing in severity for a time, after which it gradually diminishes and finally disappears entirely. A very graphic description of the results of starvation was written by Antonio Vitali, a magistrate under the First Republic, who was condemned to death, but decided to die from starvation so as to escape the ignominy of execution. He died twenty-seven days after his fast began, but for several days before this all discomfort had disappeared. Two days before the end he wrote :[5] 'La faim ne me tourmente plus. La soif a complètement cessé. L'estomac et les intestins sont tranquilles. La tête sans nuage ; la vue claire ; en un mot un calme universel règne non seulement dans mon cœur et dans ma conscience, mais encore dans tout mon organisme.' Corresponding with this it may be assumed that the movements of the empty stomach increase at first, but as the condition of the nervous system becomes more and more depressed for want of nutrition, the movements gradually diminish and finally cease.

Perhaps the contracted condition of the abdominal muscles,

which may in extreme cases give rise to a scaphoid shape of abdomen, assists in the production of the sense of emptiness, just as the stretched abdominal wall may be a factor in the production of the sense of fulness.

Section of the vagi in animals does not as a rule result in any diminution in appetite, the quantity of food taken after the operation being sometimes indeed excessive (Leuret and Lassaigne, Sédillot[6]). This is probably due to a diminution in the sense of satiety; it does not prove that the local sense of hunger is unaffected, as food continues to be taken even when the sympathetic as well as the vagal nerve-supply to the stomach is divided, the general sensation of hunger apparently remaining unaltered in both cases.

It is easy to prove that the whole of the sensation of hunger does not consist in the local sensation of emptiness. When an individual has starved for a prolonged period, a small quantity of food or water is sufficient to overcome the local discomfort, and it does this so quickly that it is clear that the relief depends upon the filling of the stomach alone, as insufficient time elapses for any appreciable quantity to be absorbed. But the general feeling of malaise remains, a much larger quantity of food being required to satisfy the hunger of the starved tissues than that of the empty stomach. For the former digestible and nutritious food is required, for the latter bulk is of primary importance. This is well seen in patients with a fistula of the upper part of the small intestine. Tiedemann had heard of such cases, and I have myself observed one. The patient was constantly hungry, although he ate enormous quantities of food. His stomach always felt full, but the general sensation of hunger remained, as most of the food escaped from the fistula with the result that the tissues continued to be starved. Relief can only be obtained in such cases by introducing food into the intestine through the distal opening of the fistula.

As the general sensation of hunger depends upon the requirements of the tissues, it is increased when these are excessive owing to abnormally active metabolism, as in diabetes and some cases of Graves' disease. Conversely the

low plane of metabolism which occurs in myxœdema is accompanied by anorexia, in spite of which the patient gains in weight. When excessive metabolism is due to a toxæmia, which also depresses the central nervous system, the effect of the latter outweighs the former, and anorexia instead of bulimia occurs. This is the case in acute fevers, tuberculosis and cancer.

Two classes of anorexia may be distinguished. In the one due to gastric disorders, such as chronic gastritis, both the local and general sensation of hunger is absent, and the allied but distinct sensation of appetite is also lost. In such conditions the local feeling of hunger probably disappears owing to the cessation of the spontaneous movements of the empty stomach caused by the local disease. In the other class of anorexia, due to a depressed condition of the central nervous system, such as occurs in neurasthenia and many other disorders, the general sensations of hunger and appetite are lost, but the local sensation of emptiness may remain unaltered. A very small quantity of food is, however, sufficient to allay the uncomfortable feeling of emptiness; as the general sensation of hunger, which in normal individuals would lead to more food being taken, is absent, nothing more is eaten.

The sensation of emptiness experienced during a period of starvation appears to be partly situated in the intestines as well as in the stomach, and the vague uncomfortable sensation, felt after the bowels have been unusually completely emptied by a purgative or enema at a period when the small intestines are empty, is in part constituted by a feeling of emptiness, which is most adequately relieved by a good meal. The sensation of emptiness in the intestines, like the corresponding sensation in the stomach, can best be explained as a result of periodical motor activity in an organ, which is abnormally excitable as a result of the absence of the normal stimuli for an unusually long period. The borborygmi, which are heard when hunger is felt, appear to originate chiefly in the small intestines; they are doubtless produced by peristalsis acting upon the mixture of gas with the digestive juices, which Boldirev[3] showed

are spontaneously secreted during the periods of motor activity.

[1] N. Tiedemann: *Physiologie des Menschen*, iii, 22. Darmstadt, 1836.

[2] W. Nicolai: *Ueber die Entstehung des Hungergefühls.* Inaug. Dissert., Berlin, 1892.

[3] *Vide* I. P. Pavlov: *The Work of the Digestive Glands.* English translation by W. H. Thompson, 2nd edition, p. 191, 1910.

[4] G. Glücksmann: *Deutsche med. Woch.*, xxxvii, 590, 1911.

[5] Quoted by G. H. Roger: *Alimentation et Digestion*, p. 131. Paris, 1907.

[6] Quoted by H. Milne Edwards: *Leçons sur la Physiologie*, xiii, 492, 1878.

CHAPTER VI

PAIN

Although as long ago as 1753 Haller[1] had failed to obtain any evidence that the pleura, peritoneum, lungs, liver, spleen or kidneys of animals were sensitive to pain, it has until recent years been regarded as self-evident that the sensory phenomena of disease could only be explained on the assumption that the viscera were well supplied with nerve-fibres, which could convey impulses leading to the sensation of pain. Since the introduction of the method of performing colostomy in two stages, the fact that the manipulation and cutting of the colon during the second stage of the operation, which is performed without an anæsthetic, produced no sensation of any kind first threw doubt upon this view. This doubt was much strengthened by the observations of Lennander[2] and James Mackenzie.[3] Lennander found on numerous occasions during operations, performed on human beings after subcutaneous injection of cocaine without a general anæsthetic, that the normal stomach, small intestine, colon and appendix could be touched with cold or hot objects, burnt with a caustic, clipped with forceps, or cut with a knife without producing any sensation. The inflamed intestine and the exposed mucous membrane of the cæcum were equally insensitive. The other abdominal viscera and the mesentery and omentum were also insensitive, but the parietal peritoneum and subserous connective tissue were very sensitive to painful stimuli, slightly to excessive heat, but not to cold or pressure. This sensibility was greatly increased even by a slight degree of inflammation. Lennander was therefore led to formulate the theory that all pain in visceral disease results from inflammation of or traction on the parietal peritoneum and its subserous connective tissue. The latter is richly supplied with fibres from cerebro-

spinal nerves, which can, according to Lennander, convey pain, whilst the viscera themselves are supplied only by the autonomic nervous system, which is not, according to him, able to do so. He believed, for example, that colic is due to the pull exerted by the distended intestine above an obstruction on its mesentery, or, where no mesentery is present, on the parietal peritoneum to which it is attached. Subsequently he added as another possible cause of colic the pressure exerted on the anterior abdominal wall by the erection of a coil of intestine in a condition of tetanic contraction, such as occurs above an obstruction.

Observations similar to those of Lennander were made during operations by James Mackenzie, but many of his patients had not even a local anæsthetic. He agreed with Lennander that pain never originates in the viscera themselves, and he showed that the parietal peritoneum, which Lennander had regarded as a very sensitive structure, is really as insensitive as the visceral peritoneum, its apparent sensibility being due to the extremely sensitive subperitoneal tissue. But Mackenzie rejected Lennander's theory in favour of one, which he had already suggested nine years before the appearance of Lennander's first paper.[4] He believes that stimuli reaching the central nervous system from diseased viscera cause no sensation themselves, but produce an irritable focus in the spinal cord, with the result that the normal afferent impulses reaching it from the muscles and other tissues supplied by this spinal segment give rise to painful impressions, which are referred to the peripheral tissues.

The observations of Lennander and Mackenzie lose some of their importance as a result of the work of Kast and Meltzer,[5] who showed that in animals visceral sensibility is diminished by the general effect of the subcutaneous injection of cocaine and by exposure to the air. They found, for example, that dogs, cats and rabbits appeared to suffer pain when their intestines were subjected to pressure by instruments and by the fingers introduced through a small incision in such a way that the viscera were not exposed, no cocaine having previously been injected. The pain was greater if the intestines were inflamed. On the

other hand, previous injection of cocaine or exposure to the air for a very short period abolished the sensibility of the intestines. Conclusions with regard to sensory phenomena drawn from experiments on animals are never very decisive, but the investigations of Kast and Meltzer receive some confirmation from two observations of Ritter,[6] who found that the human intestine was sensitive to pressure, if no cocaine had been injected and the examination was made immediately after the abdomen had been opened.

Although Mackenzie was the first to suggest that all pain in visceral disease is really felt in superficial structures, the idea of referred pain was not new. In the first edition of his ' Lectures on Rest and Pain ', published in 1863, Hilton [7] discussed the origin of the ' sympathetic pain ', which he described as occurring on the surface of the body in association with derangements of internal and often remotely situated viscera. He suggested that the superficial pain is due to some central connexion of the visceral sympathetic nerves with the peripheral sensory nerves, a connexion which was subsequently more closely studied by Verdon.[8] The following year a paper was published by Lange,[9] which foreshadowed much of the subsequent work of Ross, Head and Mackenzie, but as it appeared in Danish it was completely neglected. Lange expressed his belief that the pain which occurs in diseases of internal organs is chiefly reflex, being localized in external parts, where it is often associated with cutaneous hyperæsthesia. He suggested that the afferent impulses from the viscera pass to the spinal cord, from which they radiate along sensory tracts to the abdominal wall, to which the mind projects the sensation. In 1888 Ross [10] investigated the question afresh and came to the conclusion that two forms of pain—splanchnic and somatic—occur in visceral disease. He explained the existence of somatic pain in much the same way that Hilton had explained it under the name of sympathetic pain : impulses produced by irritation of the peripheral terminations of the splanchnic nerves are conducted by the posterior roots to the posterior horns of the spinal cord, where they ' diffuse to the roots of the corresponding somatic nerves, and thus cause an associated

pain in the territory of distribution of these nerves '. Subsequently Head [11] and James Mackenzie further developed these ideas, and as a result of their investigations the conception of referred pain—the sympathetic pain of Hilton and the somatic pain of Ross—became generally accepted. Head studied particularly the areas of cutaneous hyperalgesia occurring in visceral disease, and showed that they are identical with the areas, which receive their sensory nerve-fibres from the spinal segments, to which the afferent fibres from the diseased viscera pass. Mackenzie demonstrated that in visceral disease the muscles and parietal subperitoneal tissue supplied by the corresponding spinal segments are tender as well as the skin, and he came to the conclusion, already mentioned, that all pain in diseases of the abdomen originates in peripheral structures, in contrast to Ross, who believed in splanchnic (or visceral) and somatic (or referred) pain, and to Head, who believes that the viscera are endowed with ' deep and protopathic sensibility in a low degree ', but that the chief pain in visceral disease is referred.

The experimental and clinical investigations I am about to describe lead to the conclusion that the view held by Ross is correct, and that disease of the alimentary canal may cause true visceral pain, which is often present alone, but may be associated with pain, which is referred to the skin, muscles and connective tissues connected with the same spinal segments.

In drawing the conclusion that the viscera are insensitive, Lennander and Mackenzie did not take into consideration the fact that a nerve-ending may be sensitive to one form of stimulation—the adequate stimulus—but insensitive to all others. Thus the adequate stimulus for the eye is light, for the ear sound, and so on, although in some cases there may be several varieties of adequate stimulus. All that was proved by Lennander and Mackenzie was that the viscera are insensitive to the particular stimuli which they employed. These stimuli were cutting, pinching and pricking, none of which occur under natural conditions. It still remained possible that the viscera would be found sensitive were the adequate stimulus employed.

From a consideration of the common causes of visceral pain it might be expected that tension on the fibres of the muscular coat would prove to be the adequate stimulus, and the observations which I shall presently record confirm this view. A certain degree of distension leads to the sensation of fulness, if the internal pressure is slowly raised; the same distension rapidly produced leads to the sensation of pain.

Bayliss and Starling[12] showed that stimulation of any part of the intestine leads to contraction above and relaxation below; this reaction they called the 'law of the intestine'. It is, however, only part of a general law affecting the whole alimentary canal, contraction in one place being always associated with relaxation just below. This is seen most typically at the cardiac, pyloric, ileo-cæcal and anal sphincters, which normally relax when peristaltic waves coming from above reach them. Meltzer[13] called this law the 'law of contrary innervation', and pointed out how it applies to all hollow viscera; he attempted to explain all forms of colic, including the paroxysmal pain produced in pyloric obstruction, to a disturbance in the law of contrary innervation. I believe that his explanation is correct, and I believe, moreover, that other forms of visceral pain, such as the characteristic pain of gastric and duodenal ulcer, are due to the same cause. In colic an abnormally strong peristaltic wave occurs in one part of the alimentary canal; the part immediately below, which should normally relax, is unable to do so owing to organic disease or to spasm due to direct or reflex irritation; the segment between the peristaltic contraction and the point of obstruction is subjected to steadily increasing tension as it is shortened by the advance of the former; distension being the adequate sensory stimulus, pain is produced.

I believe that tension is the only cause of true visceral pain. Disease in the alimentary canal is often accompanied by pain due to its extension to the parietal peritoneum and sensitive subperitoneal tissue, to drag upon its peritoneal attachments as suggested by Lennander, and to reference to peripheral structures in accordance with the views of

Hilton, Lange, Ross, Head and Mackenzie. But it is generally possible to analyse the sensory symptoms in such a manner that the presence of true visceral pain can be recognized at some stage in the disease. In many conditions, such as the common forms of colic, visceral pain is present alone. On the other hand, referred pain, which is a result of the arrival in the spinal cord of impulses from an internal organ, is comparatively rarely present alone, as the impulses from the internal organ generally themselves give rise to visceral pain. But pain originating in the peritoneum in connexion with diseases of the alimentary canal is not uncommon in the absence of visceral pain.

Even when the adequate stimulus is employed, the viscera are very much less sensitive than the skin to painful stimulation. This is doubtless due, as Langley [14] has pointed out, to the small number of afferent nerve-fibres with which they are supplied, the total number distributed to the whole of the viscera of the cat being about the same as the number present in a single posterior spinal root.

One of the arguments most frequently used by those who do not believe that pain can originate in the viscera themselves is its inaccurate localization. But seeing that painful stimuli cannot be accurately localized in the skin of such parts as the back of the trunk, it is not surprising that localization is inaccurate in the viscera, which are supplied with an infinitely smaller number of nerve-fibres. According to Mackenzie, the situation of the pain of a gastric ulcer, if produced in the ulcer itself, should move when the stomach is caused to move up and down by deep respiratory movements, but it remains stationary. This argument is fallacious, as the brain learns to locate the seat of sensations according to the average position of the part in which they originate, and it is quite impossible for it to recognize changes in position of the part from the character of the sensation, as the impulses which produce this sensation are identical wherever the part may happen to be at the moment. Thus I have found that when a movable area of skin is drawn out of its usual position and some part of it is touched or pinched, the sensation produced is

referred to the point in space where the stimulated part would normally be, although, if the skin is unusually elastic, this may be several inches away from its actual position. This is exactly analogous to an interesting observation, which Mackenzie regarded as a proof that the viscera are insensitive and that all pain in visceral disease is referred to external tissues : during an operation performed without an anæsthetic the patient felt spasms of pain, which he described as arising near the umbilicus, although they were found to be due to peristaltic waves occurring in a loop of small intestine, which had been drawn out of the abdomen and was lying ten or twelve inches away from the umbilicus.

This ' average localization ' of visceral pain explains why it is most accurate in the viscera which move least, such as the gall-bladder, œsophagus, duodenum, ascending and descending colon and rectum, least accurate in the very mobile small intestines, and intermediate in the stomach. If an organ, which has become dislocated, is subsequently the seat of a painful disease, the pain is still felt in the average position and not in the new position. Thus the pain felt in cases of gastric ulcer is in the same situation whether the stomach is in the normal position or has dropped so low that the lesser curvature may be situated below the umbilicus. In a patient with a painful gall-bladder associated with a large liver the pain was felt in the exact situation which the gall-bladder must have occupied before the enlargement of the liver occurred.

Visceral pain often appears to the sufferer to be so deeply situated that it is impossible to believe that it really originates in structures such as the skin and muscles of the abdominal wall, whereas pain produced by disease of these structures feels definitely superficial. Mackenzie answers this argument by pointing out that the pain in herpes zoster may feel as if it were deeply situated, although, being a result of disease of a posterior root ganglion, it must be a referred pain. But Langley [15] has shown that the nerve cells of the afferent sympathetic neurons are situated in the posterior root ganglia, inflammation of which can therefore lead to pain referred to the distribution of the sympathetic

as well as to that of the ordinary spinal sensory nerves. The deeply situated pain in herpes zoster is in fact analogous to the abdominal pain, which occurs in disease of the spine or meninges involving the posterior roots of the lower dorsal nerves.

The visceral sensations of fulness and pain probably result from excessive stimulation of the afferent nerves, which normally convey the unfelt afferent impulses concerned in various reflexes. Both Foster [16] and Sherrington [17] have pointed out how improbable it is that the viscera should be supplied with pain fibres which have no other function, for a man may suffer from pain in a particular organ only once in the course of his lifetime. If the pain were conveyed by special nerve-fibres, it would be difficult to explain how such a special mechanism could have originated, and, when once originated, how it could have been preserved through several generations, in which it was never used, in order to give rise to a specific sensation on the one occasion in the individual's lifetime, on which he was attacked by a painful disease of the organ in question.

It is unlikely that the mucous membrane of the alimentary canal contributes to the sensation of fulness or pain, as it is only stretched when the part is greatly dilated, whereas these sensations are felt in the contracted organ when it is subjected to the adequate tension. Moreover, Professor Langley tells me that most of the afferent nerve-fibres of the intestines end in the muscular coat, medullated fibres being relatively rare in Meissner's plexus, which supplies the mucous membrane and submucous coat. Possibly the stretching of the connective tissue produced by rapid and extreme distension of the stomach or of a part of the intestine gives rise to a sensation of pain; this would be analogous to the pain, which is apparently produced by tension on the connective tissue capsule of the liver, spleen and kidneys, when these organs undergo a rapid and considerable increase in volume.

(*a*) **Oesophageal pain.**—Rapid inflation of the balloon used in the experiments on the sensation of fulness produced in the œsophagus a sensation of pain, which was slowly

replaced by a sensation of fulness without any accompanying fall in pressure. It is impossible to draw a definite line between the two sensations, the one merging into the other and every intermediate sensation between obvious fulness and obvious pain being produced according to the speed of inflation, the degree of inflation being of much less importance. The pain was localized in front as accurately as that of fulness, and was felt to be deeply situated in the middle line. It was invariably associated with a sensation of pain at exactly the same level behind, this sensation feeling as if it were situated less deeply than the anterior pain. The posterior sensation of pain, but not the anterior sensation, often radiated laterally, in one case equally in both directions, in another to the right only. The anterior sensation was one of painful fulness, as if a 'hard crust of bread had become lodged and could not be forced either upwards nor downwards'; the posterior sensation 'resembled a stitch'. The pain was in no case associated with either deep or superficial referred tenderness.

In cases of œsophageal obstruction, pain may be felt in addition to the sensation of fulness and obstruction. It is, however, a much less common symptom; whilst a sense of fulness or obstruction was only absent in one among Lamy's 134 cases, pain was absent in 60 per cent. In half the cases the pain occurred on swallowing; in the other half it was present constantly, though it was sometimes aggravated by swallowing. When it was constantly present, it was probably due to invasion of neighbouring tissues and not merely to distension as in the other cases. The pain is almost always felt behind as well as in front, and it may radiate to the sides, especially to the left, but I have never met with a case in which radiation of the sensation of fulness or obstruction occurred.

I believe that the pain felt deeply beneath the sternum is situated in the œsophagus itself, and that the pain felt in the muscles of the back, which may radiate to either or both sides, is an example of referred pain.

Professor Langley tells me that his experiments indicate that the sensory nerve-supply of the œsophagus is mainly

vagal. This agrees with our observations on the patient with bulbar palsy, in whom the sensation of fulness was only produced by a much greater rise in pressure than that required in a normal individual, and in whom no sensation of pain occurred as a result of rapid distension. Corresponding with these facts, the most characteristic referred pain from the œsophagus is the brow-ache felt by many people when they swallow an ice. This must clearly be a result of the sensory impulses ascending from the lower end of the œsophagus, with which the ice is longest in contact, by the vagi to the medulla, where they increase the irritability of a part of the neighbouring nucleus of the fifth nerve, with the result that pain is referred to its peripheral distribution. This explanation is in accordance with the opinion expressed by Gaskell[18] that the vagus contains the visceral branches of a primitive series of nerves, whose somatic sensory roots are in the sensory portion of the trigeminal nerve. The œsophagus being supplied with comparatively few sympathetic fibres, referred cutaneous and deep hyperalgesia are very rare in œsophageal disease. In our inflation experiments they never occurred, and I have never observed them in cases of obstruction. The pain sometimes felt in the muscles of the back in association with the deep substernal pain, when the œsophagus is rapidly inflated, and in cases of obstruction, in which the surrounding tissues are not involved, is, as I have said, probably a referred pain, the afferent impulses from the œsophagus in these cases being conveyed by sympathetic fibres to the segment of the spinal cord, which supplies the sensory nerves to the muscular and other tissues, in which the referred pain is felt.

(*b*) **Gastric pain.** — I have already described how slow inflation of the stomach leads to a sensation of fulness in the epigastrium when the intragastric tension reaches 12 or 15 mm. of mercury. Rapid inflation, as, for example, when carbon-dioxide is set free in the stomach after solutions of sodium bicarbonate and of tartaric acid have been successively swallowed, gives rise to pain, varying in severity with the intragastric tension, which depends upon the volume of gas produced and the tone of the muscular coat. When the

tension slowly diminishes owing to relaxation of the tone, the pain disappears. The rise in the intragastric pressure, which takes place under natural conditions, is generally so gradual that nothing more than an uncomfortable feeling of fulness results. When, however, fermentation is exceptionally active, the rise in intragastric pressure may be sufficiently rapid to produce a sensation of painful fulness in the epigastrium. In cases of organic pyloric stenosis, a tetanic contraction of the whole of the filled stomach, lasting for a few seconds, may occasionally be seen and felt, in addition to the more familiar visible and palpable peristalsis. This condition is always accompanied by violent pain, which is clearly the result of the tension on the muscular fibres produced by the enormous rise in the general intragastric pressure.

It is, however, much more common for pain to result from distension of a part of the stomach than from distension of the whole organ. In order to understand how an isolated segment can be distended apart from the rest of the stomach, it is necessary to consider in some detail the normal process of gastric peristalsis, as it has been revealed by observations under natural conditions with the X-rays.[19] Each peristaltic wave begins as a constriction near the middle of the body of the stomach, which deepens as it slowly moves towards the pylorus. About one inch from the entrance to the pyloric canal it reaches such a depth that part of the pyloric vestibule becomes almost completely separated from the rest of the stomach. The part thus cut off then diminishes in size owing to the further progress of the peristaltic wave, with the result that some of its contents are forced through the pyloric canal, the remainder being forced back as an axial reflux stream into the stomach. The waves increase in strength as digestion proceeds, and continue without intermission from immediately after the commencement of a meal until the stomach is empty. As a result of his investigations carried out in Pavlov's laboratory, Edelmann [20] concluded that hydrochloric acid stimulates peristalsis, the activity of which is proportional to the amount of acid present. He believes, moreover, that the stimulating action of chemical

substances, such as alcohol and the extractives of meat, depends upon their power of calling forth a secretion of gastric juice. The activity of peristalsis also varies with the consistence of the food ; this depends partly on the quantity of cellulose and other indigestible material present, partly on the amount of softening which takes place in cooking, and partly on the extent to which further subdivision and softening result from chewing.

The pylorus in common with all other sphincters possesses a neuro-muscular mechanism, by virtue of which it relaxes on the arrival of each peristaltic wave. But even when relaxed for the passage of chyme into the duodenum, the canal is so narrow that either no shadow of bismuth passing through it is cast by the X-rays, or, as I have occasionally observed, a very fine line joins the gastric and duodenal shadows. Some force is therefore required to press even finely-divided particles through the pylorus. Sufficient force could not be exerted by peristalsis, were it not that the waves are so deep that they shut off the extreme pyloric end of the stomach almost completely from the rest of the organ ; otherwise they would simply produce a slight rise in the general intragastric pressure, which would be no greater at one end of the stomach than the other.

The relaxation of the pyloric sphincter on the arrival of a peristaltic wave is inhibited by certain reflexes. The most important of these depend upon the presence in the duodenum of hydrochloric acid (Pavlov [21]), and the presence in the stomach of anything which might injure the duodenum owing to its chemical, mechanical or thermal properties (Cannon,[22] Joh. Müller [23]).

The internal pressure at the extreme pyloric end of the stomach, which is required to force chyme through the relaxed pylorus, is normally insufficient to give rise to any sensation. When the peristaltic waves are for any reason exceptionally strong, they begin nearer the fundus than usual ; as they also separate the pyloric end of the stomach from the rest of the organ at a point more distant from the pylorus than under natural conditions, a larger mass of chyme is compressed as each wave advances. At the same

time the greater strength of the peristaltic contraction offers a more powerful resistance to the axial reflux stream of the portion of the chyme which does not pass through the pylorus. For these reasons abnormally strong peristalsis causes an excessive rise in the internal pressure in the pyloric end of the stomach, and the tension on its muscular fibres gives rise to pain.

A second factor, which may cause an excessive rise in pressure, is absence of the relaxation of the pylorus on the arrival of a peristaltic wave; very little chyme can then escape into the duodenum, the greater part being forced back into the stomach. Such absence of relaxation may be due to the various causes already mentioned or to an organic stricture of the pylorus. The latter has the further effect of acting as a stimulant to peristalsis, in accordance with the general law that the motor activity of a muscular tube is increased in the segment above an obstruction.

Barclay's [24] X-ray observations, which are confirmed by my own more recent investigations, show that the tone and peristalsis of the stomach are quite independent functions and may be affected independently of each other in pathological conditions. Peristalsis may be excessive in a dilated atonic stomach, and much less frequently it may be deficient in a hypertonic stomach. Pain due to excessive pressure in the pyloric end of the stomach is most likely to occur when peristalsis is abnormally active in a hypertonic stomach; if at a later stage in the disease atony supervenes, peristalsis remaining unaltered, pain becomes a less prominent symptom, as the peristaltic contractions are then only able to separate the distal segment of the dilated stomach completely from the rest of the organ at a point comparatively near the pylorus.

From these general considerations I believe that it is possible to explain the origin of all true gastric pain, as distinct from referred pain and the pain which is associated with adhesions and with extension of the disease from the stomach to the parietal peritoneum.

The pain of the acute indigestion, which results from a rapidly eaten and insufficiently chewed meal, is due to the

association of increased peristalsis with inhibition of pyloric relaxation caused by the presence of hard particles of food. Such particles remain in the stomach until all the fluid and semi-fluid contents have left, so that more time is allowed for the gastric juice to soften them and for peristalsis to break them up. Even at the end these processes are often incomplete, and very strong peristalsis is finally required to force the particles through the pylorus. It is only when the stomach has finally been emptied in the natural way or by vomiting that relief is experienced.

The excessive peristalsis and the rigidity of the pylorus in cases of organic obstruction account for the severe pain which may occur in this condition. It begins soon after a meal has been taken, and increases in severity as peristalsis becomes more active on account of the secretion of hydrochloric acid in non-malignant cases and the formation of organic acids in cases of pyloric carcinoma. The increase in the tone of the stomach, which occurs as it empties itself, is an additional factor in the progressive aggravation of the pain, as it makes it possible for the powerful peristaltic waves to shut off the pyloric end of the stomach more completely. In some cases, indeed, the pain only begins two or three hours after a meal, when the stomach has evacuated part of its contents. The pain may last for many hours, and in severe cases, in which evacuation by the natural passage is never complete, it is only relieved by vomiting or lavage.

Gastric pain is frequently associated with the presence of excess of free hydrochloric acid in the stomach. This is a comparatively rare occurrence apart from organic disease, the pain and hyperacidity being generally associated with gastric and duodenal ulcer, but occasionally with gall-stones and chronic appendicitis. In spite of the doubts which have from time to time been expressed on the subject, the recent extensive investigations by Willcox,[25] Craven Moore [26] and other English physicians on cases, in which the diagnosis was confirmed by operation, prove beyond question that an excessive quantity of free and of active hydrochloric acid is present in the large majority of cases of gastric and

duodenal ulcer. In an earlier chapter I described the observations which led us to conclude that the contact of free hydrochloric acid with the intact mucous membrane and with the surface of an ulcer does not itself cause pain. But the relief afforded by alkalies suggests that the pain is in some way connected with the presence of free hydrochloric acid. I believe that this apparent contradiction is due to the fact that the stimulating effect of the acid on peristalsis and the abnormally prolonged inhibition of pyloric relaxation, which occurs when the hyperacid chyme reaches the duodenum, can only cause pain when food is present in the stomach, as no rise in internal pressure can occur when there is nothing in the stomach upon which the muscular coat can contract. Hyperacidity is particularly likely to cause the excessive motor activity which results in pain when ulceration is present, as it may be presumed that the acid stimulates the afferent nerve-endings more strongly when they are exposed in an ulcer than when the mucous membrane is intact. Moreover, a greater number of nerve-fibres pass from the deep structures of the wall of the stomach, which are exposed by ulceration, than from the mucous membrane. For the same reasons the stimulating effect of alcohol and of coarse particles of food on peristalsis is exaggerated. The afferent impulses which pass from the surface of the ulcer are insufficient in themselves to give rise to any sensation, but they produce reflexes which may result in pain, increased peristalsis occurring in the case of a gastric ulcer and inhibition of pyloric relaxation in the case of a duodenal ulcer. This view is confirmed by the relief given to the pain of gastric ulcer by paralysing the afferent nerve-endings in the ulcer by means of orthoform. My X-ray observations show that the presence of a duodenal ulcer has the additional effect of increasing the tone of the muscular coat of the stomach; as I have already pointed out, the association of hypertonus with excessive peristalsis and inhibition of pyloric relaxation is very favourable for the production of pain.

True pylorospasm may perhaps be an additional factor in the production of pain in gastric and duodenal ulcers which

involve the pyloric canal. The reflex spasm of the pyloric sphincter in such cases would be analogous to the spasm of the anal sphincters, which occurs in cases of anal ulceration. It would lead to a rise in tension within the pyloric part of the stomach by increasing the resistance to the passage of chyme through the pylorus, as this is probably not completely arrested when relaxation of the sphincter is inhibited so long as its tone is not increased.

Frequently, but by no means invariably, an ulcer near the cardia gives rise to pain immediately after meals and an ulcer near the pylorus about an hour and a half to two hours after meals, intermediate situations being associated with intermediate times. The time of onset of pain in duodenal ulcer is much more constant; it almost always begins between two and three hours after a meal. The time relations can be explained by considering at what moment free hydrochloric acid comes into contact with the ulcer so as to lead to an exaggeration of the reflex motor efforts which produce pain. The hydrochloric acid is secreted mainly by the glands of the proximal two thirds of the stomach, the secretion of the extreme pyloric end being actually alkaline. As no peristalsis and consequently no churning of the contents occur in the fundus, the outer layer of chyme remains constantly very acid. A cardiac ulcer is therefore bathed in acid gastric juice at a very early stage in digestion. The food which first reaches the pyloric end of the stomach is alkaline; it is only after a considerable interval that the acid gastric juice reaches this part to any great extent, and, as peristalsis is constantly active in the pyloric part, the gastric juice is greatly diluted by the large quantity of food with which it is mixed. Consequently an hour or more may pass before there is sufficient free acid to irritate an ulcer near the pylorus. The intermediate portion of the stomach differs from the pyloric end in secreting an acid juice; this does not, however, remain in contact with the mucous membrane in a concentrated state as it does in the cardiac end, for it is constantly mixed by peristalsis with the alkaline food. Consequently an ulcer in this situation is irritated by acid at an interval after

a meal intermediate between that which elapses in cardiac and in pyloric ulcers.

In a series of cases of duodenal ulcer examined with the X-rays, I have always found that the stomach begins to empty itself immediately after the food has been swallowed and that the evacuation is at first rapid. When the pain begins between two and three hours after a meal, only a small proportion of the food is still present in the stomach, and the hypertonic condition constantly present in cases of duodenal ulcer reaches its greatest development, owing to the increase in tone which occurs as the bulk of the gastric contents diminishes. Under these conditions peristaltic contractions can produce a complete separation of the pyloric part from the rest of the stomach at a considerable distance from the pylorus. Owing to the excessive and prolonged secretion of normal gastric juice, which is the cause of the so-called hyperchlorhydria of duodenal ulcer, the proportion of gastric juice and of hydrochloric acid in the chyme increases as digestion proceeds. At first most of the acid combines with the alkaline salts and the proteins of the food, and the small quantity of free acid which reaches the duodenum is rapidly neutralized by the alkaline intestinal juice, bile and pancreatic juice, so that the relaxation of the pylorus is only occasionally inhibited. But after two or three hours, the proportion of acid present being greater, some of it reaches the ulcer before it is neutralized. The inhibition of pyloric relaxation, which the contact of acid with the intact duodenal mucous membrane produces, is exaggerated by the presence of the ulcer, so that the peristaltic waves advance against a pylorus, which only opens at considerable intervals in order to permit the passage of a small quantity of hyperacid chyme into the duodenum. Immediate relief to the pain follows the administration of alkalies or proteins, which neutralize the acid, or of food or water, which dilutes it; relief is also produced by vomiting and lavage, which remove the acid and at the same time empty the stomach so that nothing is left upon which the muscular coat can contract. The pain disappears spontaneously only when the stomach has become completely empty. This generally occurs about

an hour after the onset of pain, but if the evacuation of the stomach is hindered by partial obstruction due to cicatrization or inflammatory swelling round the ulcer, the pain lasts for many hours. As the sense of fulness is produced by exactly the same mechanism as pain, when the stimulus is less powerful, a sensation of fulness is generally felt between two and three hours after food for some months before the first occurrence of pain. At a later stage pain may be replaced by this sensation after some meals, and, as Moynihan[27] has pointed out, the characteristic hunger pain is preceded and accompanied by a sensation of fulness, distension or weight in the same situation. I have already explained how the patient often erroneously ascribes this to flatulence and repeatedly tries to eructate, temporary relief being at last obtained by the return of some of the air swallowed in the preceding unsuccessful attempts. The salivation which often occurs when the pain is most severe is an additional cause of the aërophagy.

Bertrand Dawson[28] has pointed out that the most frequent exception to the common time relations occur in gastric ulcers situated very near the pylorus, the pain often beginning immediately after food. This is doubtless due to the inflammatory swelling round the ulcer causing partial obstruction, so that the pain is produced in the same manner and with the same early onset as it is in cases of cicatricial or cancerous pyloric obstruction.

Occasionally there is no excess of hydrochloric acid in gastric ulcer. But as alkalies still give relief, it may be assumed that the pain is due to the reflex results of irritation of the ulcer by acid, although the latter is not present in more than the usual proportion. In the rare cases in which there is actually deficient gastric secretion, the ulcer is always indurated and fixed by adhesions; the pain is then mainly extra-gastric in origin, but it may perhaps be due in part, as suggested by Alexis Thomson,[29] to spasm and increased peristalsis resulting from the interference with the motor functions of the stomach.

So far I have only referred to the effect of gastric and duodenal ulcers on the peristalsis of the pyloric end of the

stomach and on the pyloric sphincter. These effects are most marked when an ulcer is situated near the pylorus, and consequently ulcers in the cardiac end and in the body of the stomach often give rise to no pain until they have reached the peritoneal surface and led to adhesions or local peritonitis. But such ulcers may cause true visceral pain in other ways than those already described. Cannon[30] has shown that relaxation of the cardia is inhibited by the contact of free hydrochloric acid with the mucous membrane of the cardiac end of the stomach. The inhibition is increased by the presence of an ulcer in its neighbourhood, just as the inhibition of pyloric relaxation is increased by the presence of an ulcer in the duodenum. Consequently towards the end of a meal, when free hydrochloric acid is present, the cardia does not relax as each bolus reaches it; œsophageal peristalsis becomes exaggerated, and pain produced by distension of the extreme lower end of the œsophagus is felt high up in the epigastrium before the meal is finished. In confirmation of this, Barclay[26] has shown with the X-rays that ulceration near the cardia may give rise to such serious functional obstruction of the œsophagus that dilatation ensues. In a patient, who died after the performance of gastrostomy for a supposed stricture of the lower end of the œsophagus, a very small ulcer was found near the cardia; the œsophagus was greatly dilated, but there was no obstruction.

In organic hour-glass contraction of the stomach the stricture leads to exactly the same motor disturbances in the proximal part of the organ that pyloric obstruction causes in the distal part. Peristalsis, which is normally absent from the fundus, can now be observed with the X-rays, and tetanic contraction of the whole of the proximal segment can occasionally be seen and felt. Excessive peristalsis causes pain by the tension exerted on the part of the stomach just above the obstruction, and tetanic contraction causes pain by the tension exerted on the whole of the contracting segment.

X-ray observations have shown that an ulcer in the body of the stomach may cause a local spasmodic contraction,

which leads to a temporary and partial hour-glass constriction (Jollasse,[31] Barclay,[24] Jonas[32]). This does not by itself cause pain, as it can often be seen with the X-rays when the patient is quite comfortable. The absence of pain is due to the fact that such a spasmodic contraction never leads to peristalsis or tetanic contraction in the proximal part of the stomach; this is in fact an important aid in the distinction of functional from organic hour-glass contraction. When, however, cicatrization of the ulcer has produced partial obstruction, the ulcer, if not completely healed, may cause a local spasm, which makes the obstruction complete. In such cases peristalsis and tetanic contraction may occur in the proximal part of the stomach and give rise to pain.

It is often difficult to distinguish the gastric symptoms of cholelithiasis and chronic appendicitis from the symptoms of gastric and duodenal ulcer. This is due to the fact that the pain is produced in the pyloric end of the stomach by exactly the same mechanism, although the primary cause is different. Thus Lichty [33] has shown how frequently excess of hydrochloric acid is present in the stomach in diseases of the gall-bladder and biliary ducts; in a considerable proportion of his cases, the gastric analyses were made because there were definite symptoms of gastric disturbance associated with gall-bladder symptoms, and in some the former were present alone and the disease of the gall-bladder was only discovered at the subsequent operation. Similarly many cases of chronic appendicitis with predominant gastric symptoms have been shown to be associated with hypersecretion of gastric juice. In both cases the hyperacidity causes abnormally active peristalsis. The experiments of Cannon and Murphy [34] show that injury to the intestines leads to inhibition of pyloric relaxation. Cholelithiasis and chronic appendicitis have probably the same effect, as in spite of the excessive activity of peristalsis there is often a slight delay in the evacuation of the stomach. I have observed, moreover, that these conditions may lead to gastric hypertonus. Excessive peristalsis being associated with hypertonus and inhibition of pyloric relaxation,

pain is produced, exactly as it is in cases of ulceration. This view is confirmed by the fact that the contracted pyloric end of the stomach can occasionally be felt as a tumour and can often be seen during operations in cases of duodenal ulcer, gall-stones and chronic appendicitis. The condition has been called 'pylorospasm', but Mr. Moynihan agrees with me that the term is not strictly accurate, as it is not the pyloric sphincter itself, but the pyloric vestibule which is abnormally contracted.

In an earlier chapter I pointed out that when the stomach is distended with gas, the sensation of fulness is felt deeply beneath the upper part of the anterior abdominal wall in the middle line. The sensation of pain produced by rapid distension of the whole stomach with gas and that produced by tetanic contraction in pyloric obstruction is felt in the same situation; it varies in different individuals, but as a rule extends from near the xiphisternum to near the umbilicus. When only part of the stomach is subjected to abnormal tension, pain is felt in the upper or lower section of this area, according to whether the pain originates in the cardiac or pyloric division of the stomach. In a case of hour-glass stomach, upon which Mr. R. P. Rowlands operated, the contraction was due to the cicatrization of an ulcer close to the cardia, the proximal segment being no larger than a hen's egg; in this case the pain was felt very high in the epigastrium. In the more common cases of hour-glass stomach, in which the constriction is near the centre, the pain extends further downwards. On the other hand, the pain in ulcers of the pyloric end of the stomach and of the duodenum and in pyloric obstruction is felt nearer the umbilicus, the exact situation varying in different individuals. The gastric pain in cholelithiasis and chronic appendicitis, being produced by the same mechanism, is felt in the same region. Pain is occasionally felt a little to the left of the middle line in gastric cases; it is sometimes felt to the right in cases of duodenal ulcer, especially when excessive tension also occurs in the proximal end of the duodenum owing to partial obstruction caused by cicatrization, inflammatory swelling or spasm.

It was formerly thought that the pain in gastric ulcer was felt exactly over the situation of the ulcer. Anatomical and radiographic observations have shown, however, that this is not the case. I have already pointed out that the sensation produced in a movable organ must be felt in an average position, as the adequate stimulation of any organ or part of an organ can only produce a single sensation, which is referred to the same situation, whatever the position of the organ at the moment may be. Moreover, the pain of a gastric or duodenal ulcer is not produced in the ulcer itself, but in a part of the stomach which is subjected to abnormal tension owing to the reflexes resulting from irritation of the ulcer; consequently, even if the stomach were fixed and the power of localization were as accurate as it is in the œsophagus, the pain would not be felt over the ulcer itself.

The pain produced by pressure in the epigastrium in gastric disorders was until recently regarded as a result of direct pressure upon the stomach; when the tenderness was localized to a small area, it was supposed that this area corresponded with the situation of some local disease such as an ulcer. More accurate comparison between the situation of the tender area and that of the ulcer as revealed post mortem and at operations, the fixity of the tender area when the stomach changes its position as a result of a deep inspiration or of an alteration in posture, and the fact, first disclosed by the X-rays, that the tender area is generally situated entirely outside the area occupied by the stomach showed that this view was incorrect. I have already described how some of these arguments were used to support the untenable theory that spontaneous pain does not originate in the stomach itself. But as the tenderness of an organ can only be revealed by direct pressure upon it, these facts prove beyond doubt that the pain produced by pressure in the epigastrium in gastric disorders can rarely be due to tenderness of the stomach itself. This is indeed what would be expected from a consideration of the adequate stimulus to gastric pain. Pressure could only give rise to pain by increasing the tension in a part of the stomach,

in which it was already excessive. Corresponding with this, on the few occasions on which I have observed tetanic contraction of a part of the stomach in hour-glass constriction or of the whole stomach in pyloric obstruction, the pain was increased by pressure on the tumour formed by the contracted organ; such pressure must have the effect of increasing the internal tension. Occasionally also, when peristalsis is visible and palpable, pressure near the pylorus increases the pain, and in the rare cases in which the contracted pyloric part of the stomach forms a palpable tumour, this is generally found to be tender. But pressure applied over a gastric ulcer could not possibly cause local pain, as ulcers in the alimentary canal are insensitive to mechanical stimuli.

In the large majority of cases the epigastric tenderness is a result of the irritable focus in the spinal cord produced by abnormal impulses reaching it from the stomach; pressure on the skin, muscles and connective tissue supplied by the irritable spinal segment produces an exaggerated sensory effect. This is generally most marked in the muscles, for cutaneous hyperalgesia is often absent, and deep pressure always causes more pain than pressure applied as far as possible to the skin alone. That the connective tissues are also concerned is shown by the pain produced by pressure over the linea alba in patients in whom the recti muscles are widely separated. Jonas[35] has pointed out that in gastric ulcer pressure produces much less pain when the recti are voluntarily contracted than when they are relaxed. This suggests that some structure more deeply situated than the abdominal muscles is also tender in these cases. Roux's[36] anatomical investigations have shown that the solar plexus is directly beneath the common area of deep tenderness in gastric disorders, and most Continental authorities have accepted his conclusion that this is the actual seat of the deep tenderness. But the sensory sympathetic nerve-fibres have their cell station in the posterior root ganglia and pass through the solar and other plexuses without undergoing any structural change; as the sensation produced by irritation of afferent nerve-fibres is always referred to their peripheral distribution, pressure on the

solar plexus—if it had any effect at all—would give rise to no local sensation, but to pain in the viscera. Moreover, there is no evidence that nerve-fibres are more sensitive to direct pressure when they are conveying impulses than when they are resting. The sensitive structure which lies deeper than the abdominal muscles is, therefore, neither the stomach itself nor the solar plexus; it is highly probable that it is the subperitoneal tissue, which Ramström[37] has shown is very richly supplied with sensory nerve-endings.

Referred tenderness is always greatest when spontaneous pain is greatest, as the spinal segment is then most irritable owing to the arrival of impulses from the affected part. In some cases, especially in men, tenderness may be present only during the period in which pain is felt; in other cases, especially in neurotic and anæmic women, it is continuous in the intervals between the attacks of pain, and it may last to a less extent for a considerable period after all spontaneous pain has disappeared.

It was at one time thought that cutaneous hyperalgesia and localized deep tenderness were characteristic symptoms of gastric ulcer. But the knowledge gained from the frequent abdominal operations of recent years has shown that this view is incorrect. On the one hand, cutaneous hyperalgesia occurs not infrequently in the functional dyspepsia of anæmic and neurasthenic patients, as Bertrand Dawson[38] has shown and my own experience confirms, and deep tenderness occurs even more frequently among the same class of patients, although, indeed, it is unusual to find it localized to a small area except in organic disease. On the other hand, cutaneous hyperalgesia is frequently absent in gastric and duodenal ulcer, especially in men; I have, in fact, only observed it in a single case of duodenal ulcer in which the diagnosis was confirmed by operation, and in that case it was situated on the left side in the situation characteristic of gastric disease. Deep tenderness may also be absent, but this is comparatively rarely the case if the patient be examined when the pain is at its worst.

These facts are not surprising when the cause of cutaneous and deep hyperalgesia is considered. On the one hand, the

same irritable focus in the spinal cord is produced by excessive afferent impulses passing from the stomach, whatever their primary cause may be ; on the other hand, an irritable focus is particularly prone to occur in neurasthenic and anæmic individuals, but it is only produced by exceptionally strong stimuli in patients with a healthy nervous system.

When the disease of the stomach has given rise to local inflammation of the parietal peritoneum, pressure over the inflamed area gives rise to pain, for Mackenzie and Lennander have shown that pressure is an adequate stimulus to the inflamed subperitoneal tissue. Consequently in such cases the exact locality of the disease can be recognized. As the tenderness is due to inflammation, it is constantly present and is largely independent of spontaneous pain.

Professor Langley has shown that the movements and rise of blood pressure, which occur in lightly anæsthetized animals when any of the white rami communicates or any of the strands of abdominal sympathetic nerve-fibres are stimulated, are not observed on stimulating the vagus at the lower end of the œsophagus, although respiratory and other reflexes, indicating the presence of afferent fibres, are obtained. He concludes that painful sensations in the stomach and small intestines are conveyed by sympathetic and not by vagal nerve-fibres. This corresponds with the fact that referred pain and tenderness in gastric disease occur almost invariably in the distribution of the seventh, eighth and ninth dorsal segments, as was first clearly demonstrated by Head, and very rarely in the distribution of the trigeminal nerve, as would occur if the afferent impulses were conveyed by the vagus. Moreover, the gastric crises of tabes, in which the character of the pain is exactly similar to that produced by primary gastric disease, must result from irritation somewhere in the course of the nerve-fibres which convey painful sensations from the stomach ; and various surgeons, acting upon Foerster's[39] suggestion, have shown that the crises can be abolished by section of the posterior roots of the seventh, eighth and ninth dorsal nerves.

Miller[40] has recently proved that the reflexes caused by irritation of the gastric mucous membrane depend on afferent

impulses, which are conveyed by vagal and not by sympathetic nerve-fibres. The afferent vagal fibres from the stomach, which never convey painful sensations, end therefore in the insensitive mucous membrane, whereas the afferent sympathetic fibres, which can convey painful sensations, end in the sensitive muscular coat.

(c) **Intestinal pain.** Using the same apparatus as that employed in our experiment on the sensation of fulness, Mr. Digby and I found that rapid inflation resulted in a sensation of pain both in the colon and the rectum. The rectal pain was accompanied by a sensation of tenesmus, especially when the ampulla was inflated. The pain, like the sensation of fulness, was produced instantaneously and slowly disappeared, unless the air was suddenly allowed to escape, in which case it disappeared at once.

I have already described our observations on a patient in whom a loop of the pelvic colon had prolapsed through the gap left by the removal of part of the sacrum ; they showed that the sensation of fulness is visceral in origin and due neither to tension on the peritoneum nor to pressure on the abdominal wall. At the same time the pain produced by rapid distension was shown to originate in the intestine.

True intestinal pain only occurs in those diseases in which there is abnormal motor activity. Catarrhal colitis may give rise to a feeling of fulness owing to the distension produced by the excessive development of gas, but there is no acute pain. Acute colitis without ulceration is only accompanied by pain when the occurrence of severe diarrhœa shows that the motor activity of the intestine is excessive. In the absence of diarrhœa, ulceration causes no pain so long as the peritoneum is not involved and cicatrization has not resulted in obstruction. Thus typhoid fever and tuberculous ulceration are frequently unaccompanied by any pain, and simple ulcerative colitis and dysentery may also run a quite painless course. Ulcers of the colon can be touched with a probe or painted with silver nitrate solution through a sigmoidoscope without giving rise to any sensation, and the frequent absence of all pain throughout the disease

shows that the mere contact of the intestinal contents, however irritant they may be, does not cause pain.

Peristalsis is unable by itself to cause pain. It is only when the onward passage of the intestinal contents is prevented by organic or spasmodic obstruction that the adequate rise in internal pressure is produced. Thus colic accompanying the diarrhœa caused by chemical or mechanical irritation of the intestines, whether associated with acute colitis or not, is probably due to the simultaneous occurrence of violent peristalsis of one segment and stricture due to the spasmodic contraction of a more distal segment, the intervening bowel being thus subjected to a sudden considerable rise in internal pressure. Spasmodic contractions are more likely to occur as reflexes from an ulcerated surface than from the unbroken mucous membrane, although the afferent impulses, which are produced by the contact of the irritating intestinal contents with the ulcer and which give rise to the reflexes, do not directly cause pain. In many cases of colitis pain only occurs after meals, and in other cases the pain is aggravated by the taking of food. This is due to the fact that the consumption of food is the most powerful of all stimuli to the motor activity of the colon, as I have been able to prove by means of X-ray observations on normal individuals and on patients with colitis.

The most violent form of intestinal pain is the colic which occurs in cases of organic obstruction. As I have already pointed out, this is produced by the pressure exerted on the muscular coat of the bowel above the obstruction by the intestinal contents, which are forced forward by a violent peristaltic wave or a tetanic contraction and are prevented from passing onward by the obstruction. When a tetanic contraction occurs in a segment of intestine, the latter can be felt to rise under the hand as a hard tumour and the patient complains of great pain ; the intestine becomes once more impalpable as the paroxysm of pain disappears. I do not think that a tetanic contraction of the empty intestine can by itself cause pain. When the intestine is distended with fluid fæces and gas, they are squeezed out by the contracting segment, so that no great rise in the internal

pressure occurs. If, however, the passage of fæces and gas in one direction is prevented by an organic obstruction, then the rise in pressure necessary to produce pain occurs. This is the cause of the severe paroxysms of pain which accompany the tetanic contraction of the colon above a growth.

Pain is a frequent symptom of the enterospasm, which is the cause of the spastic constipation occurring in neurotic individuals, either alone or associated with the passage of membranes composed of coagulated mucus in muco-membranous colitis. In these conditions the contraction of the bowel is present for considerable periods instead of occurring in short paroxysms as in cases of organic obstruction. The pain is not due to the tonic contraction alone, as the contracted colon can often be felt for several weeks after treatment has led to the complete disappearance of the pain and constipation. Moreover, the empty colon of normal individuals is sometimes so contracted as a result of the tone of its muscular coat that part of it can be felt—especially in the left iliac fossa—as a solid cord, although pain and constipation are absent. When, however, the tonic contraction involves a segment of large intestine containing solid fæces, the tension on its circular muscle-fibres may be sufficient to produce a sensation of uncomfortable fulness or even of slight pain. The severer attacks of pain are probably due to exactly the same cause as ordinary intestinal colic, the further progress of the intestinal contents forced forward by a peristaltic wave being prevented by the tonically contracted condition of the bowel, so that they distend the intervening portion and give rise to pain; or the tension on the wall of the contracted colon may be increased by more fæces being forced into it. Consequently the pain is relieved when further distension is prevented by the removal of the contents of the colon by an enema, although the actual passage of the fæces and water through the contracted bowel may give rise to a sufficient increase in pressure to cause considerable pain. The pain in lead colic is probably due to a similar combination of peristalsis and spasm to that which occurs in severe cases of spastic constipation.

Uncomplicated intestinal pain is only accompanied by true visceral tenderness when it is a result of enterospasm or of tetanic contraction occurring above a growth. In these conditions pressure upon the contracted intestine increases the interna ltension to the minimal adequate height or aggravates any pain which is already present. The adequate internal tension is particularly likely to be attained if the pressure be directed against a rigid structure such as the spine or the iliac fossa. It is so easy in cases of this sort to recognize the contracted colon by palpation that it is impossible to doubt that the pain produced by pressure is true visceral pain. Moreover, equally firm pressure on the abdomen in the immediate neighbourhood of the tender colon produces no pain.

I have already pointed out that visceral pain is accurately localized in fixed viscera and inaccurately in very movable viscera. While therefore pain in the very mobile small intestine is felt near the umbilicus and pain in the movable parts of the colon between the umbilicus and pubes, pain in the duodenum and the last few inches of the ileum, in the ascending and descending colon, at the hepatic, splenic and pelvi-rectal flexures, and in the rectum, is felt in regions which correspond with a fair degree of accuracy with the true positions of these parts. Thus obstruction in the small intestine generally causes pain in the neighbourhood of the umbilicus, but obstruction at the extreme end of the ileum, due to cancer of the cæcum or adhesions following appendicitis, leads to attacks of pain, which may begin near the umbilicus, but culminate in the right iliac fossa, as the last few inches of the ileum, which constitute its least movable part, are then distended. Obstruction at the splenic flexure causes paroxysms of pain, which often begin below the umbilicus when the central and most movable part of the transverse colon is the seat of painful stimulation, but generally end in the neighbourhood of the obstruction when the terminal and least movable part of the transverse colon is subjected to excessive internal tension. Similarly attacks of pain in cancer of the rectum often begin in the middle line just above the pubes and end in the neighbourhood of the

sacrum or in the rectum itself, where they produce a sensation of painful tenesmus.

The pain of appendicitis is in part due to distension of the appendix itself. This true appendicular pain is felt in the neighbourhood of the umbilicus. A large proportion of acute attacks begin with pain in this situation, and a history is frequently obtained of previous short and less severe attacks, in which the pain was confined to the centre of the abdomen. If the appendix is removed at a sufficiently early stage in acute appendicitis, there may be no spontaneous pain in the right iliac fossa; the appendix is then found to be distended with inflammatory products, but the inflammation has not reached the parietal peritoneum.

Pressure upon a distended appendix must increase the internal tension and so give rise to pain. I have been able to demonstrate this in eight patients, in whom the inflamed appendix was situated in the pelvis. No great tenderness was observed on abdominal palpation, but on rectal examination the appendix could be felt and rolled under the finger, extreme pain being thereby elicited, although the examination was otherwise almost or quite painless. As the examination was made *per rectum*, there could be no question of the pain being due to pressure on the parietal peritoneum or on hyperalgesic skin or voluntary muscle. The diagnosis of an inflamed pelvic appendix, uncomplicated by spreading peritonitis, was in each case confirmed by operation.

In the presence of peritonitis, however localized it may be, pain is generally felt in the right iliac fossa, the common history of pain beginning near the umbilicus and then settling in the cæcal region being obtained. This secondary pain is due to the peritonitis and originates in the parietal peritoneum and not in the appendix. In cases of appendicitis, in which the appendix is not situated in its common position, the initial pain begins as usual near the umbilicus, but the secondary peritoneal pain is felt in the actual situation of the disease and not in the right iliac fossa, corresponding with the fact, experimentally demonstrated by Lennander, that the power of localizing peritoneal pain is comparatively accurate. Thus I have seen two cases in which the cæcum

was situated immediately below the liver and the appendix was adherent to the gall-bladder; in both cases the pain began near the umbilicus and finally settled in the situation of the peritonitis, but it was at no period present in the right iliac fossa. When an appendix situated in the pelvis or in the retrocæcal fossa becomes inflamed, the pain begins near the umbilicus, but continues respectively in the pelvis and in the loin. The true appendicular pain often disappears with the onset of peritoneal pain, as it is the result of distension and can therefore no longer occur after perforation and collapse of the distended appendix.

Wherever the appendix may be situated, the skin and muscles over the right iliac fossa are often the seat of referred tenderness and sometimes of referred pain. The constant situation of the referred tenderness and pain is due to the sympathetic nerve-supply of the appendix being always the same, whatever abnormality there may be in its situation. It has been suggested that the deep tenderness is due to pressure on the irritable ileo-cæcal plexus, as Keith [41] showed that the ileo-cæcal sphincter is situated directly beneath MacBurney's point. But the arguments, which prove that the deep epigastric tenderness in gastric diseases is not due to pressure on the solar plexus, apply here with equal force. MacBurney's point is simply the centre of the area of referred muscular hyperalgesia.

The true appendicular pain situated in the centre of the abdomen is often present before any referred pain or tenderness. When peritonitis is present it gives rise to tenderness, which is due to the inflammation of the subperitoneal tissue. With peritonitis in the right iliac fossa, it is impossible to judge how much of the deep tenderness is due to peritonitis and how much to referred hyperalgesia of the various structures of the abdominal wall. When the peritonitis is confined to the pelvis or other remote situation, the tenderness in the right iliac fossa is all due to referred hyperalgesia, the situation of the peritoneal tenderness giving an indication of the localization of the appendix.

The experiments of Langley and the observations of Head on referred cutaneous hyperalgesia indicate that the sensa-

tion of pain in the intestines is conveyed by sympathetic nerve-fibres through the posterior roots of the tenth and eleventh dorsal nerves to the spinal cord. This conclusion is confirmed by the observations of Bruns and Sauerbruch [42] on a tabetic patient, in whom they had divided the posterior roots of the seventh, eighth and ninth dorsal nerves for gastric crises. Although the latter completely disappeared, an attack of acute indigestion, brought on by drinking excess of sour milk three months after the operation, was accompanied by pain in the lower part of the abdomen, considerably below the situation of the pain of the crises. I have already pointed out that the fibres conveying visceral sensations probably ascend the spinal cord in the posterior columns. The viscera may therefore occasionally be anæsthetic in tabes, and a case of acute appendicitis may end fatally without pain, tenderness or rigidity, although, as in a case recorded by Conner,[43] the patient's mind remains alert until the end.

According to Langley, the pelvic nerves, which originate from the first three sacral nerves, convey painful sensations from the rectum. Corresponding with this, referred pain in diseases of the rectum is felt in the skin and deep tissues supplied by the first, second and third sacral segments of the spinal cord.

[1] A. v. Haller: *Opera Minora*, i, 356, in the edition published at Lausanne, 1763.

[2] K. G. Lennander: *Centralbl. f. Chir.*, xxviii, 209, 1901 (first paper), and *Journ. Amer. Med. Ass.*, xlix, 836, 1907 (last paper). *Vide* also E. Nyström: *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, xxi, 125, 1910.

A summary of James Mackenzie's views is contained in his book *Symptoms and their Interpretation*, London, 1909.

[4] J. Mackenzie: *Med. Chron.*, xvi, 293, 1892, and *Brain*, xvi, 321, 1893.

[5] L. Kast and S. J. Meltzer: *Mitteil a. d. Grenzgeb. d. Med. u. Chir.*, xix, 586, 1909.

[6] C. Ritter: *Centralbl. f. Chir.*, xxxv, 609, 1908.

[7] J. Hilton: *Lectures on Rest and Pain*, pp. 64 and 74, 1863.

[8] H. W. Verdon: *St. Thomas's Hosp. Reps.*, v, 286, 1874.

[9] Lange: *Neuralgier og deres Behandling, Hospitalstid*, 1875. Quoted by K. Faber: *Deutsch. Arch. f. klin. Med.*, lxv, 338, 1899.

[10] J. Ross: *Brain*, x, 350, 1888.

[11] H. Head: *Brain*, xvi, 1, 1893, and xvii, 339, 1894.

[18] *Vide* E. H. Starling: *Recent Advances in the Physiology of Digestion*, London, 1906.

[19] S. J. Meltzer: *Arch. f. Verdauungskrankheiten*, ix, 450, 1903.

[14] J. N. Langley: *Brain*, xxvi, 23, 1903.

[15] J. N. Langley in Schäfer's *Physiology*, ii, 687, 1900.

[16] M. Foster: *Text-book of Physiology*, iv, 1891.

[17] C. S. Sherrington in Schäfer's *Physiology*, ii, 990, 1900.

[18] W. H. Gaskell: *Journ. of Physiology*, vii, 1, 1886.

[19] *Vide* A. F. Hertz: *Quarterly Journal of Medicine*, iii, 373, 1910.

[20] Edelmann: Dissertation, St. Petersburg, 1905, quoted by R. Magnus: *Ergebnisse der Physiol.*, vii, 30, 1908.

[21] I. P. Pavlov: *The Work of the Digestive Glands*. English translation by W. H. Thompson, 2nd edition, p. 187, 1910.

[22] W. B. Cannon: *Amer. Journ. of Physiology*, xx, 283, 1907.

[23] Johannes Müller: *Zeitschrift f. diät. u. physik. Ther.*, viii, 585, 1905.

[24] A. E. Barclay: *Brit. Med. Journ.*, 1908, ii, 712.

[25] W. H. Willcox: *Quarterly Journal of Med.*, iii, 93, 1909.

[26] F. Craven Moore: *Proc. Roy. Soc. Med.*, iii, Medical Section, p. 25, and Surgical Section, p. 111, 1910.

[27] B. G. A. Moynihan: *Duodenal Ulcer*, p. 101. London, 1910.

[28] Bertrand Dawson: *Brit. Med. Journ.*, 1908, i, 1088.

[29] Alexis Thomson: *Brit. Med. Journ.*, 1909, i, 648.

[30] W. B. Cannon: *Amer. Journ. of Physiology*, xxii, 105, 1908.

[31] O. C. Jollasse: *Fortschr. a. d. Geb. d. Röntgenstr.*, xi, 1906. Quoted by S. Jonas, loc. cit. 32.

[32] S. Jonas: *Wien. klin. Rundschau*, Nr. 47–48, 1909.

[33] J. A. Lichty: *Amer. Journ. of the Med. Sciences*, cxli, 72, 1911.

[34] W. B. Cannon and F. T. Murphy: *Annals of Surgery*, xliii, 512, 1906.

[35] S. Jonas: *Verhandl. d. Kongr. f. inn. Med.*, xxv, 445, 1908.

[36] J. Ch. Roux: *Revue de Méd.*, xix, 878, 1899.

[37] M. Ramström: *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, xviii, 314, 1908.

[38] Bertrand Dawson: *Brit. Med. Journ.*, 1905, ii, 1032.

[39] O. Foerster and H. Küttner: *Beiträge z. klin. Chir.*, lxiii, 245, 1909. *Vide* also O. Bruns and F. Sauerbruch: *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, xxi, 173, 1910.

[40] F. R. Miller: *Journ. of Physiology*, xlii, 409, 1911.

[41] *Vide* F. Treves: *Brit. Med. Journ.*, 1902, i, 1592.

[42] O. Bruns and F. Sauerbruch: *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, xxi, 173, 1910.

[43] L. A. Conner: *Journ. Amer. Med. Ass.*, lv, 1427, 1910.

CHAPTER VII

VARIATIONS IN THE SENSIBILITY OF THE ALIMENTARY CANAL

The sensibility of the alimentary canal, like that of the skin, varies in different healthy individuals. I have already pointed this out in connexion with our experiments on the sensibility of the alimentary canal to heat and cold; the intestines, for example, are generally insensitive to thermal stimulation, but in a small proportion of people they are sensitive. We also found considerable individual variations in the minimal internal pressure required to produce a sensation of fulness in all parts of the alimentary canal, and rapid inflation caused pain with greater ease in some individuals than in others. These variations did not appear to depend in any way upon the mental development of the individuals examined.

Apart from the differences in the sensibility of the viscera in normal individuals, the possibility of pathological variations must be considered. It has been suggested that the pain associated with hyperchlorhydria is sometimes a result of hyperæsthesia of the gastric mucous membrane. But I have not yet met with a patient in whom pain or other sensation was produced by the introduction of 0·5 per cent. hydrochloric acid into the empty stomach.

I believe, however, that it is possible for an individual to become hypersensitive to the stimulus, which results in the sensations of fulness and pain. It is a well-recognized fact that the sensibility of the skin and the special senses can be increased by practice, and there is no reason why it should not be possible to develop visceral sensibility in a similar manner. When an individual concentrates his attention on every slight sensation connected with his digestive organs, he appears to acquire an increased sensibility,

which results in a lowering of the threshold of consciousness and hence of the minimal adequate stimulus, and in an exaggerated sensory response to stimuli which were already adequate. This is probably the explanation of the symptoms of abdominal hypochondriasis. I have already pointed out how easy it is to misinterpret visceral sensations, even when the individual concerned has a good knowledge of physiology and pathology. When a hypochondriac has not this knowledge, and when his viscera have become hypersensitive in the manner described, the sensations are often seriously and grotesquely misinterpreted, with the result that the patient may end by becoming insane. Thus a hypochondriacal patient paying close attention to the sensation produced in his œsophagus when hot or cold food is swallowed and to the disappearance of the sensation as the food enters the stomach, comes to the conclusion that his gullet ends in a blind sac. Another with a hypertonic stomach and an increased sensibility to the adequate stimulus interprets the sensation of fulness, which comes after a very little food has been eaten, as an indication that all nourishment should be refused because the passage is blocked.

In some cases the sensations felt in the abdomen are not true visceral sensations at all, but are purely mental in origin, as in the case I described in my first lecture of a patient whose symptoms seemed to point to a thermal hyperæsthesia, but in whom the stomach and intestines were shown to be completely insensitive to hot and cold stimulation.

The *faiblesse irritable* of the nervous system, which is characteristic of neurasthenia and which also occurs in chlorosis, results in the same lowering of the minimal adequate stimulus and exaggeration of the sensory response which occur in hypochondriasis. Consequently digestive processes, which are normally imperceptible, become perceptible, and conditions, which would otherwise lead to nothing more than a sensation of slight discomfort, give rise to pain. Owing to the irritability of the central nervous system, afferent impulses also give rise to an exaggerated reflex motor and secretory response, which accounts for the

frequency with which the constipation of neurasthenia is complicated by enterospasm and muco-membranous colitis ; the resulting hypertonic condition of the colon makes it possible, as I have already pointed out, for a moderate degree of fæcal retention to produce a sensation of fulness or pain.

In visceroptosis the afferent impulses, produced in the erect position by the constant drag of the viscera on the sensitive structures, to which their peritoneal supports are attached, give rise to no sensation if the patient is otherwise healthy. But if he is also neurasthenic, discomfort and pain result owing to the low minimal stimulus required to produce sensation.

Owing to the excitable condition of the nervous system in neurasthenia and in chlorosis, the spinal segment which receives painful impulses from a diseased organ becomes exceptionally irritable, and the irritability spreads to neighbouring spinal segments, with the result that areas of cutaneous and deep tenderness are more constantly present and are considerably more extensive in neurasthenic and anæmic patients than in otherwise normal individuals suffering from similar gastric disorders.

CONCLUSIONS

THE following conclusions may be drawn from the experimental and clinical investigation of the sensibility of the alimentary canal in health and disease.

1. The mucous membrane of the alimentary canal from the upper end of the œsophagus to the junction of the rectum with the anal canal is insensitive to tactile stimulation.

2. The mucous membrane of the œsophagus and the anal canal is sensitive to thermal stimulation, but that of the stomach and intestines is insensitive.

3. The mucous membrane of the œsophagus and stomach is insensitive to stimulation by dilute hydrochloric acid and dilute organic acids, and the rectum, but not the anal canal, is insensitive to stimulation by glycerine. Contact of alcohol with the mucous membrane of all parts of the alimentary canal gives rise to a sensation of heat.

4. The surface of gastric and intestinal ulcers is no more sensitive to tactile, thermal and chemical stimulation than the intact mucous membrane.

5. The sensation of fulness in the alimentary canal is due to a slow increase in the tension exerted on the fibres of its muscular coat; the adequate tension is constant for each segment, but the volume of contents necessary to produce this tension varies with the tone of the muscle-fibres.

6. The sense of fulness in the rectum has a special character, by virtue of which it produces the call to defæcation.

7. Hunger consists in a general sensation of malaise and weakness in the body as a whole and a local sensation of emptiness in the abdomen. The latter is due to the periodical motor activity of the stomach and intestines during fasting, in which a condition of muscular hypertonus and nervous hyperexcitability exists.

8. The only immediate cause of true visceral pain is tension; this is exerted on the muscular coat of hollow organs and on the fibrous capsule of solid organs. The sensation of pain

in the alimentary canal is due to a more rapid or greater increase in tension on the fibres of its muscular coat than that which constitutes the adequate stimulus for the sensation of fulness.

9. Pain in diseases of the alimentary canal is most frequently true visceral pain ; it is sometimes due to spread of the disease to surrounding sensitive structures or to tension exerted on the peritoneal connexions ; it may also be situated in the skin, muscles and connective tissues, to which it is referred from the segment of the central nervous system, which receives the afferent nerves from the affected organ.

10. Tenderness in diseases of the alimentary canal is most frequently due to hyperalgesia of the skin, voluntary muscles and connective tissues supplied by the segment of the central nervous system, which receives the afferent nerves from the affected organ. It may also be due to the increase in tension within the organ produced by the external pressure giving rise to the adequate stimulus of visceral pain ; this is rare in the stomach, but comparatively common in spasmodic conditions of the colon and in appendicitis. Lastly, it may be due to the spread of the disease to the parietal peritoneum.

11. Visceral sensibility is exaggerated by training in hypochondriasis, and visceral and referred sensations are exaggerated by the irritable condition of the central nervous system in neurasthenia and anæmia.

INDEX OF AUTHORS

INDEX

OXFORD: HORACE HART
PRINTER TO THE UNIVERSITY